IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

MARILYN F. QUIRIN, as Executor of the Estate of RONALD J. QUIRIN, Deceased,	)	
Plaintiff,	)	Civil Action No. 13-cv-02633
v.	)	Judge Joan B. Gottschall
LORILLARD TOBACCO COMPANY, *et al.*,	)	
Defendants.	)	

DEFENDANT UNION CARBIDE CORPORATION'S MOTION *IN LIMINE* TO PRECLUDE DR. CARL BRODKIN FROM TESTIFYING THAT EXPOSURE TO UNION CARBIDE ASBESTOS WAS A CAUSE IN FACT OR A "SUBSTANTIAL FACTOR" IN CAUSING PLAINTIFF'S MESOTHELIOMA

Pursuant to Fed. R. Evid. 702 and *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993), Defendant Union Carbide Corporation ("Union Carbide") respectfully moves the Court, *in limine*, for an order precluding Plaintiff's causation expert, Dr. Carl Brodkin, from offering expert testimony or opinion that Mr. Quirin's alleged exposure to Union Carbide Calidria asbestos in joint compounds[1] participated in causing his mesothelioma or was a "substantial factor" in bringing it about.

I. INTRODUCTION

Dr. Carl Brodkin is expected to testify at trial that Mr. Quirin's alleged exposure to Union Carbide Calidria asbestos caused his mesothelioma merely because, as Dr. Brodkin puts it, "[i]t's the aggregate dose [of asbestos] that increases the risk and causes the disease." Dec. 17, 2012 Dep. Tr. at 239:16-17 (attached as Exhibit 1) ("Brodkin Dep."). This theory—that *all* exposures

[1] The facts of this case, and Union Carbide's participation in the asbestos business more generally, are set forth in Union Carbide's Motion for Summary Judgment and related pleadings.

to asbestos contribute to causing mesothelioma because each exposure contributes to an individual's aggregate asbestos *dose*— has been exposed by numerous courts as "inadmissible speculation that is devoid of responsible scientific support." *Smith v. Ford Motor Co.*, 2013 WL 214378, at *2 (D. Utah Jan. 18, 2013) (attached as Exhibit 2). Indeed, a Washington state court precluded Dr. Brodkin from offering *the very same* causation opinions that Plaintiff seeks to admit here, finding that his practice of extrapolating "assessment[s] of causation in a particular case" from the purported "dose-response" relationship that exists generally between asbestos exposure and *risk* of developing mesothelioma "is not a sound scientific methodology." *Free v. Ametek et al.*, No. 07-3-04092-9 SEA (Wash. Super. Ct Feb. 29, 2008), at 5 ("*Ametek* Order") (attached as Exhibit 3).[2] Courts in numerous jurisdictions have now excluded similar opinions in asbestos litigation as fundamentally at odds with prevailing legal standards for establishing causation. *See Smith*, 2013 WL 214378, at *5.[3]

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[2] *Ametek* was decided under Washington law, which continues to apply the "general acceptance" standard set forth in *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). Nevertheless, the *Ametek* court's findings regarding the unscientific nature of Dr. Brodkin's causation opinions are equally applicable in a *Daubert* context. Accordingly, Union Carbide has attached a copy of the *Ametek* Order to this Motion for the Court's review.

[3] The *Smith* court cited the following cases: *Betz v. Pneumo Abex*, LLC, 44 A.3d 27 (Pa. 2012); *In re Toxic Substances Cases*, 2006 WL 2404008 (Pa. Com. Pl. Aug. 17, 2006) (attached as Exhibit 4); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. Ct. App. 2011); *Moeller v. Garlock Sealing Techs., LLC*, 660 F.3d 950 (6th Cir. 2011); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765 (Tex. 2007); *Smith v. Kelly–Moore Paint Co., Inc.*, 307 S.W.3d 829 (Tex. App.—Ft. Worth 2010); *Georgia-Pacific Corp. v. Stephens*, 239 S.W.3d 304 (Tex. App.—Houston [1st Dist.] 2007); *Lindstrom v. A-C Prod. Liab. Tr.*, 424 F.3d 488 (6th Cir. 2005); *Wills v. Amerada Hess Corp.*, 379 F.3d 32 (2d Cir. 2004). Testimony based on the "every exposure" theory of causation was also rejected in *Bartel v. John Crane*, 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004); *In re W.R. Grace & Co.*, 355 BR. 462 (Bkrtcy D. Del. 2006); *Basile v. American Honda Motor Co., Inc.*, 2007 WL 712049, at *2 (Pa. Com. Pl. Feb. 22, 2007) (attached as Exhibit 5); *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 226-27 (Pa. 2007); *Moeller v. Garlock Sealing Technologies, LLC*, 660 F.3d 950, 954-55 (6th Cir. 2011); *Holcolm v. Georgia Pacific, LLC*, 289 P.3d 188, 197 (Nev. 2012); *Sclafani v. Air and Liquid Systems Corp.*, 2013 WL 2477077, at *4-5 (C.D. Cal. May 9, 2013) (attached as Exhibit 6); and *Anderson v. Ford Motor Co.*, --- F.Supp.2d ----, 2013 WL 3179497 (D. Utah June 24, 2013) (attached as Exhibit 7).

The same result should follow here. To our knowledge, no court applying Illinois law has ever held—or even suggested—that an exposure to asbestos (or any other environmental agent) can be deemed a cause in fact of a plaintiff's injury merely because the exposure contributed in some undefined manner to the plaintiff's aggregate dose, and thereby in some undefined way to the *risk* that such an injury could occur. Dr. Brodkin's methodology of substituting "risk" for "cause" thus seeks to circumvent the legal standard that Plaintiff must meet to establish legal causation, which by definition cannot assist the jury to make findings that are consistent with Illinois law. Dr. Brodkin's conclusion that Mr. Quirin's alleged exposure to Calidria asbestos "caused" his mesothelioma amounts to nothing more than *ipse dixit* disguised as expert testimony, as Dr. Brodkin admittedly "ha[s] not done a supply specific assessment" with respect to Calidria asbestos and thus relies on "no information" whatever regarding Mr. Quirin's alleged exposure to Calidria compared to his exposures to asbestos from numerous other sources—many of which, unlike Calidria, contained highly carcinogenic amphibole asbestos. Brodkin Dep. at 175:12-17; *see also id* at 41:6-15 (acknowledging that amphiboles are "several times more potent than chrysotile in causing mesothelioma").

Finally, the methodology that Dr. Brodkin relies upon in reaching his causation opinions is applied nowhere outside the courtroom in asbestos litigation, and is therefore precisely the type of "unscientific speculation offered by a genuine scientist" that is inadmissible under *Daubert*. *Segle v. Stegmiller*, 2012 WL 1570129, at *1 (N.D. Ill. May 3, 2012) (quoting *Rosen v. Ciba–Geigy Corp.*, 78 F.3d 316, 318 (7th Cir. 1996)) (attached as Exhibit 8).[4] For each of these reasons, Dr. Brodkin must be precluded from giving his causation opinions at trial.

[4] For purposes of this Motion, Union Carbide does not challenge Dr. Brodkin's training or qualifications as an expert generally. As demonstrated below, the causation opinions he has proffered in this case are inadmissible on other grounds.

II. ARGUMENT

A. Dr. Brodkin's Causation Opinion Would Not Assist The Jury To Make Factual Determinations In Accordance With Illinois Tort Law.

As a threshold matter, Dr. Brodkin's causation opinion is inadmissible because it seeks to short-cut the legal standard for causation that Plaintiff must satisfy to meet her burden of proof. Courts applying Fed. R. Evid. 702 routinely preclude experts from offering testimony that is "contrary to law," because such testimony "cannot be said to be… helpful to the trier of fact." *Loeffel Steel Prods., Inc. v. Delta Brands, Inc.*, 387 F. Supp. 2d 794, 806 (N.D. Ill. 2005); *accord Clements-Jeffrey v. City of Springfield*, 2011 WL 3207363, at *5 (S.D. Ohio July 27, 2011) (excluding expert testimony "found to be contrary to law, and thus inadmissible") (attached as Exhibit 9); *Anderson v. Dairy Farmers of Am., Inc.*, 2010 WL 3893601, at *11 (D. Minn. 2010) ("[The expert's] mitigation opinion is contrary to law and therefore would not be helpful to a finder of fact in deciding the issues in the case.") (attached as Exhibit 10).

In *Nolan v Weil-McLain*, the Illinois Supreme Court made clear that while "asbestos plaintiffs face unique challenges in showing causation," there is no "exception for asbestos cases which relieve[s]… plaintiffs from meeting the same burden as all other tort plaintiffs" and that evidentiary rules governing asbestos litigation cannot supplant "black-letter, general principles of tort causation law." 910 N.E.2d 549, 558 (Ill. 2009) (citing *Thacker v. UNR Indus., Inc.*, 910 N.E.2d 549, 558 (Ill. 2009)). Thus, *Nolan* "reaffirm[s] the axiomatic rule that a plaintiff alleging personal injury in any tort action—including asbestos cases—must adduce sufficient proof that the defendant caused the injury." *Id.* To meet this standard, Plaintiff must demonstrate that each defendant's conduct was a "cause in fact" of Mr. Quirin's injuries, *i.e.*, that it was "a material element and a substantial factor in bringing" them about. *Krywin v. Chicago Transit Auth.*, 938 N.E.2d 440, 447 (Ill. 2010). And it is well settled as a matter of Illinois law that "[c]onduct is a

material element and a substantial factor if, absent the conduct, the injury would not have occurred." *Id.*; *accord Abrams v. City of Chicago*, 811 N.E.2d 670, 675 (Ill. 2004).

Dr. Brodkin's causation opinion cannot be squared with these governing legal principles because it specifically *disclaims* any attempt to "pars[e] out" a single exposure or exposure sub-set attributable to a particular defendant and determine whether those exposures can be deemed a but-for cause of Mr. Quirin's mesothelioma. *See* Brodkin Dep. at 239:18-240:10. Nor can Dr. Brodkin "say one way or the other" whether Mr. Quirin's alleged exposure to asbestos from any particular source likely would have caused his injuries on its own, as his testimony regarding Kent brand cigarettes attributable to Defendant Lorillard Tobacco Company illustrates:

> Q So is it your opinion that Mr. Quirin's alleged Kent cigarette smoking in and of itself would not have been sufficient to cause his mesothelioma?
>
> A. I can't say that one way or the other. I don't think medical science has a way of addressing that question. Mr. Quirin's exposure to asbestos smoking the Kent micronite filters between 1954 and 1956 in my opinion is a significant exposure. I've identified it as a component part of his exposure. That exposure is not as great as the cumulative exposure he had including all occupational exposures. So the exposure to Kent micronite did increase his mesothelioma, but it's the aggregate of his exposure in the environmental as well as the occupational setting that resulted in his total risk and caused his mesothelioma.
>
> *So I have no way of parsing out a single component, whether it be Kent micronite or some other component.*

Brodkin Dep. at 239:18-240:10 (emphasis added).

Instead, Dr. Brodkin's theory appears to be that merely showing that each defendant's asbestos or asbestos-containing product contributed to Mr. Quirin's aggregate asbestos dose, thereby adding—by some unspecified amount—to his "total risk," makes that exposure a substantial causation factor. *Id*. at 240:7. But we are aware of no toxic tort case arising under Illinois law that has ever held or otherwise suggested that an undefined increase in *risk* can be

equated with factual *causation*. Nor is it appropriate to consider such a causation framework for asbestos litigation *only*. The Supreme Court reiterated in *Nolan* that Illinois law has "not carve[d] out an exception for asbestos cases which relieved those plaintiffs from meeting *the same burden as all other tort plaintiffs*." 910 N.E.2d at 558 (emphasis added). Because Dr. Brodkin's testimony would do nothing to assist the jury to decide questions of proximate cause in accordance with Illinois law, it is not "relevant to the task at hand." *Daubert*, 509 U.S. at 597.

B. Dr. Brodkin's Causation Opinion Is Improper Because It Amounts To Nothing More Than An *Ipse Dixit* Value Judgment Of How "Cause" Should Be Defined In Asbestos Litigation.

Dr. Brodkin's failure to base his opinions on *any* information specific to Mr. Quirin's alleged exposure to Calidria asbestos also ignores the well-established tenet that "expert testimony must be rejected if it lacks an adequate basis in fact." *Nunez v. BNSF Ry. Co.*, 2012 WL 2874059, at *4 (C.D. Ill. July 13, 2012) (citing *Cella v. United States*, 998 F.2d 418 (7th Cir. 1993)) (attached as Exhibit 11). As noted above, Dr. Brodkin has failed to perform any type of "specific assessment" with respect to Calidria (*see* Brodkin Dep. at 175:12-20), and he is "[a]bsolutely not" able to determine whether Calidria asbestos—or any other alleged exposure in Mr. Quirin's exposure history—actually participated in "caus[ing] his disease." *Id*. at 239:13-15; *see also id.* at 240:9-10 ("I have no way of parsing out a single component, whether it be [Mr. Quirin's exposure to crocidolite asbestos from Kent cigarettes] or some other component.").[5]

Dr. Brodkin's conclusion that *each* of these exposures can nevertheless be characterized as a "cause" appears to be based upon his surmise that because each exposure to asbestos

[5] Irrespective of any attempt by Dr. Brodkin to characterize Mr. Quirin's alleged exposures to asbestos from other sources or other defendants' products, Dr. Brodkin readily conceded that he has made no such attempt with respect to Union Carbide or Calidria asbestos. *See id.* at 175:12-17 ("Q. And so if I understand your answer correctly, sir, with respect to Calidria chrysotile, you have no information as to Mr. Quirin's alleged dose; is that correct? A. That's true. I have not done a supply specific assessment.").

contributes to an individual's aggregate asbestos dose, and "the aggregate [dose]… result[s] in [the individual's] total risk," then each exposure must participate causally, at least in cases where mesothelioma actually develops. *Id.* at 240:4-8. But that is not a scientific opinion: none of the scientific literature that Dr. Brodkin purports to rely upon stands for the proposition that mesothelioma actually develops biologically as a result of an individual's "total risk" or "aggregate dose" of asbestos, much less that an individual exposure can be deemed causal solely on the basis that it contributes to either. Rather, the opinion derives from Dr. Brodkin's own value judgment as to how the concept of "cause" should be defined in the context of asbestos litigation, and accordingly is no more scientific than an opinion claiming that each defendant "caused" Mr. Quirin's injuries because each defendant held a market share in asbestos sales during his alleged exposure period and could have been a source of his exposure—a *legal* theory of causation that has been rejected in asbestos litigation and dismissed out of hand in Illinois.[6]

The court in *Smith*, *supra*, is the latest to pull the curtain back on this attempted sleight of hand and lay bare the unscientific nature of Dr. Brodkin's methodology. There, the court found that "[w]hen carefully examined," the "all exposures" theory "is precisely the kind of testimony the Supreme Court [of the United States] observed as being nothing more than the 'ipse dixit of the expert.'" 2013 WL 214378, at *2 (quoting *Gen. Elec. v. Joiner*, 522 U.S. 136, 146 (1997)). Just as Dr. Brodkin would attempt to do here, the plaintiffs' expert in *Smith* opined that each defendant's asbestos must be "rule[d]… in" as a causal agent, "boldly stating that Mr. Smith's mesothelioma was caused by his total and cumulative exposure to asbestos, *with all* exposures and all products playing a contributing role." *Id.* at *3 (emphasis in original, internal quotations

[6] *See, e.g.*, *White v. Celotex Corp.*, 907 F.2d 104, 106 (9th Cir. 1990) (finding market share theories of liability "entirely inappropriate in asbestos litigation"); *Smith v. Eli Lilly & Co.*, 560 N.E.2d 324, 337 (Ill. 1990) (concluding "that market share liability is not a sound theory, is too great a deviation from our existing tort principles and should not be applied" in DES litigation).

omitted). But this theory "asks too much from too little evidence as far as the law is concerned," and "seeks to avoid not only the rules of evidence but more importantly [plaintiffs'] burden of proof." *Id.* Indeed, the court aptly compared the proffered opinion to

> a homicide detective who discovers a murdered man from a large family. Based on his and other detectives' training and experience the detective knows that family members are often the killer in such cases. When asked if there are any suspects the detective says he cannot rule out any of the murdered man's relatives. This would be reasonable, but it would not allow the detective to attribute legal liability to every family member on the basis of such a theory.

Id. As *Smith* makes clear, the premise that an asbestos exposure or exposure sub-set *could* have participated in causing mesothelioma by virtue of its incremental contribution to the *risk* of developing the disease cannot by itself support a finding of causation: "Just because we cannot rule anything out does not mean we can rule everything in." *Id.* at *3. Thus, the "all exposures" theory "does virtually nothing to help the trier of fact decide the all-important question of specific causation" and accordingly is inadmissible as expert testimony. *Id.* at *4.

Similarly, in *Betz v. Pneumo Abex LLC*, the Supreme Court of Pennsylvania squarely rejected the contention that an expert can draw scientifically valid conclusions about the causal role of a particular asbestos exposure by relying on the premise that "[e]ach of the exposures to asbestos contributes to the total dose that causes mesothelioma." 44 A. 3d 27, 31 (Pa. 2007).[7] The plaintiff's expert in *Betz* offered a variety of analogies in an attempt to support such an inference: that exposures act cumulatively like "marbles into [a] glass of water until the water finally overflows"; that it is impossible to discern whether "General Eisenhower" or "every troop in the field" should be credited with winning the Second World War; and that because one

[7] Although *Betz* was decided under the law of Pennsylvania, a *Frye* jurisdiction, the court cited the *Daubert* standard a number of times, with particular emphasis on its requirement that expert testimony derived from the scientific method must be properly "grounded in testing." 44 A.3d at 47; *see also id.* at 48 n.21 & n.23.

cannot be sure whether "every blow" or "just the last blow that [a] boxer took" resulted in his knock-out, it must be presumed that "the cumulative effect of all the blows would be the cause." *Id.* at 35. But while certain of these analogies could be said to be "true in a figurative and honorary fashion," the court "fail[ed] to see" how they "b[ore] any connection whatsoever to science." *Id.* at 57. Rather, the use of such analogies simply "convey[ed]" that the underlying theory they are designed to support is "inconsistent with both science and the governing standard for legal causation." *Id.*; *see also Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 539, 541 (Ga. Ct. App.), *cert. denied* (Ga. 2011) (affirming exclusion of proffered expert opinion that "[t]o the extent" the plaintiff "was exposed to any of" the defendants' asbestos-containing products, "they contributed in a cumulative fashion to his total asbestos dose, which is what caused his mesothelioma"; adopting trial court's *Daubert* ruling that such an opinion "was not the product of reliable principles and methods").

This Court should join the numerous courts cited above, and preclude Dr. Brodkin from offering expert opinions in this case that are ultimately rooted in his personal philosophy of how "cause" should come to be defined in asbestos litigation. Such opinions are "not admissible under Rule 702" because they are "purely speculative." *United States v. Vance*, 2011 WL 2633842, at *6 (N.D. Ill. July 5, 2011) (quoting *United States v. Davis*, 772 F.2d 1339, 1333-43 (7th Cir. 1985)) (attached as Exhibit 12).[8]

C. Dr. Brodkin's Causation Opinion Also Fails To Meet The *Daubert* Standard For Admission Of Expert Testimony.

At bottom, Dr. Brodkin concludes that causation can be ascribed to Calidria asbestos without any "specific assessment" of its alleged role in Mr. Quirin's exposure history because

[8] For much the same reasons, Dr. Brodkin also should be precluded from offering an opinion that Mr. Quirin's alleged exposure to Calidria asbestos was "substantial" or a "substantial factor."

"[m]esothelioma is a dose response disease" and any exposure necessarily contributed to Mr. Quirin's "aggregate dose." Brodkin Dep. at 175:16-17; 239:15-17. That is precisely the type of unscientific methodology that lacks a "reliable foundation" and should be excluded under "the gatekeeping function *Daubert* has imposed on trial judges." *Richman v. Sheahan*, 415 F. Supp. 2d 929, 932 (N.D. Ill. 2006).

The gatekeeping inquiry mandated by *Daubert* requires courts "to make a determination as a precondition to admissibility, that proffered scientific evidence rests on a reliable foundation …." *Id.* at 932 (citing *Daubert*, 509 U.S. at 589, 597). In this respect, "[t]he focus is not on the expert's conclusions, but on the underlying methodology." *Id.* at 933 (citing *Daubert*, 509 U.S. at 593-95)). In *Daubert*, the Supreme Court provided a non-exhaustive list of several factors that may "bear upon the [reliability] inquiry," including (1) whether a theory or technique can be, and has been, tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) the known or potential rate of error; (4) the existence and maintenance of standards controlling the technique's operation. 509 U.S. at 593-94. In addition, while it is no longer "the exclusive test for admissibility" of expert testimony, "general acceptance" of an expert's methodology is another factor that may bear upon the Court's reliability analysis. *See Richman*, 415 F. Supp. 2d at 933 n.4. At bottom, the "[e]ssential[ ] purpose of the rule in *Daubert* is 'to make sure that when [scientific experts] testify in court they adhere to the same standards of intellectual rigor that are demanded in their professional work.'" *Murata Mfg. Co. v. Bel Fuse, Inc.*, 2008 WL 656045, at *5 (N.D. Ill. Mar. 5, 2008) (quoting *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 318 (7th Cir. 1996)) (attached as Exhibit 13).

Dr. Brodkin's Calidria causation opinion easily fails to meet this standard because nowhere outside of the courtroom in asbestos litigation do members of the scientific community

attempt to draw reliable conclusions about the causal relationship between a disease—*e.g.*, mesothelioma—and a specific environmental factor suspected to be toxic—*e.g.*, asbestos in certain joint compounds—without first conducting a thorough assessment of the exposure and comparing it to that of other suspected causal agents. Indeed, as several courts have recognized,

> A scientifically-reliable methodology that is recommended by the World Health Organization and the National Academy of Sciences for drawing a sound conclusion as to the relationship between an individual's disease and a specific factor suspected of causing that disease entails a three-step process. This three-step process includes: (1) a determination of the plaintiff's level of exposure to the toxin in question, (2) from a review of the scientific literature, proof that the toxin is capable of producing the illness, or general causation, and the level of exposure to the toxin which will produce that illness (i.e., the dose-response relationship) must be ascertained, and (3) establishment of specific causation by demonstrating the probability that the toxin caused the particular plaintiff's illness, which involves weighing the possibility of other causes of the illness.

Parker v. Mobil Oil Corp., 16 A.D.3d 648, 651 (N.Y. App. Div. 2005). "This three-step process has been acknowledged in numerous cases as generally accepted and reliable." *Id.* (citing cases). As explained in the attached affidavit of Dr. Suresh Moolgavkar, an expert in the carcinogenicity and epidemiology of asbestos, applying a reliable methodology in mesothelioma cases requires "estimating the additional risk, if any, imposed by the exposure at issue after taking into account the probability that the disease occurred spontaneously and the probability that other exposures, including other asbestos exposures and ionizing radiation, caused the disease." *See* Moolgavkar Affidavit at ¶ 94 (attached as Exhibit 14).[9] Brodkin has done no such analysis here.

[9] The scientific methodology outlined by Dr. Moolgavkar stands in stark contrast Dr. Brodkin's method, which equates a presumed, but unproven, increase in risk associated with every exposure, regardless of dose, with substantial factor causation. Under the scientific method, causation can be inferred only by determining the fraction of a person's total disease risk attributable to an exposure. This method requires examining the *relative contribution* to dose and risk that an exposure provides – analyses that Dr. Brodkin made no attempt to undertake.

In fact, Dr. Brodkin freely admits to ignoring the first criterion identified in *Parker*—an assessment of Mr. Quirin's alleged "level of exposure" to Calidria asbestos—and his analysis sidesteps the second and third criteria as well. Instead, Dr. Brodkin simply reasons that if mesothelioma can be attributed to an individual's *aggregate* exposure to asbestos, it follows that mesothelioma can be attributed to individual asbestos exposures or exposure sub-sets on the ground that they necessarily contributed in some manner to the individual's aggregate asbestos dose. However, that proposition is in no way generally accepted in the fields of medicine, pathology, or epidemiology—or any other scientific discipline, for that matter. And to the extent such a theory amounts to anything more than Dr. Brodkin's own brand of "common sense," it certainly cannot be falsified or validated through scientific testing—the *Daubert* reliability factor that "has been recognized as the most important." *U.S. Automated Sprinkler Co. v. Reliable Automated Sprinkler Co*., 2010 WL 1266659, at *3 (S.D. Ind. Mar. 25, 2010) (citing *Chapman v. Maytag Corp.*, 297 F.3d 682, 688 (7th Cir. 2002)) (attached as Exhibit 15). Indeed, it is difficult, if not impossible, to imagine what empirical data could support—or refute—whether Dr. Brodkin's conceptualization of "cause" is the right one. This should "indicate[ ]" to the Court that Dr. Brodkin's "proffered opinions cannot fairly be characterized as scientific knowledge" and "amount to nothing more than unverified statements unsupported by scientific methodology." *Chapman*, 297 F.3d at 688.

Given the nature of Dr. Brodkin's methodology, it is not surprising that he has failed to make any type of "specific assessment" of the risk associated with Mr. Quirin's alleged exposure to Calidria in comparison with his total risk or his risk from other exposure sources. This, too, renders Dr. Brodkin's "opinion on specific causation inherently unreliable." *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1162 (E.D. Wash. 2009). Much like Dr. Brodkin

would attempt to do here, the medical expert precluded from giving testimony in *Henricksen* had opined that the plaintiff's cancer was caused by an exposure to benzene, but had never "attempt[ed] to quantify dose or even estimate Henricksen's exposure." *Id.* Rather, the expert had simply "presume[d] that exposure to benzene in gasoline can cause [the plaintiff's cancer] and that Henricksen's exposure was sufficient." *Id.* Because Dr. Brodkin's Calidria opinion rests on the very same type of impermissible presumption, "there is simply too great an analytical gap between the data and the opinion proffered." *Id.* at 1154 (quoting *Joiner*, 522 U.S. at 146).

Equally important for *Daubert* purposes is the fact that Dr. Brodkin's opinions are based on an underlying factual premise that neither the medical nor the scientific communities have ever validated: that all exposures to asbestos resulting in some non-zero contribution to an individual's aggregate asbestos dose actually increase the risk of developing mesothelioma. As the court in *Butler v. Union Carbide Corp.* held in excluding similar testimony under the federal *Daubert* standard followed by Georgia state courts, the "'any exposure' theory is, at most, scientifically-grounded speculation: an untested and potentially untestable hypothesis." 712 S.E.2d 537, 552 (Ga. App. 2011). In fact, as explained by Dr. Moolgavkar, contrary to Dr. Brodkin's no-safe-level hypothesis, "there is excellent affirmative epidemiologic evidence that exposure to low levels of chrysotile asbestos does not increase the risk of mesothelioma." *See* Moolgavkar Affidavit at ¶ 94. Dr. Brodkin fails to address this and further fails to take into account available studies showing "no evidence that exposure to joint compound increases the risk of mesothelioma." *Id.* at ¶ 61; *see also id.* at ¶ 97 (noting that Dr. Brodkin "ignores the large body of epidemiologic literature showing that exposure to low levels of chrysotile asbestos does not increase the risk of mesothelioma and does not add to the risk imposed by other exposures."). This scientific evidence is particularly relevant for assessing the causal role of Mr. Quirin's

alleged exposures to Calidria asbestos—a pure form of short-fiber chrysotile asbestos uncontaminated with tremolite or other amphibole types of asbestos. *See id.* at ¶¶ 43-52 (noting that amphibole asbestos is substantially more potent than chrysotile asbestos and explaining the problem of amphibole contamination in studies of chrysotile asbestos); *see also* Brodkin Dep. at 41:6-15. Dr. Brodkin's failure to take into consideration the relative potency differences among Mr. Quirin's various asbestos exposures in deeming them all substantial contributing factors renders his causation opinion unreliable and inadmissible. *Id.* at ¶ 97("Dr. Brodkin has not critically evaluated whether Mr. Quirin's alleged bystander exposure to chrysotile asbestos added to the substantial risk imposed by his total amphibole exposure.").

Throughout his deposition, Dr. Brodkin referred to statements and regulations promulgated by the EPA and OSHA, among other agencies, as evidence that low-dose exposures to chrysotile asbestos can be hazardous. *See* Brodkin Dep. at 151:10-21; 176:13-19. Safety regulations of this sort are "typif[ied]" by "speculation, conflicts in evidence, and *theoretical extrapolation*" by agencies acting under their prophylactic mandate to "protect[ ]… the public health." *Ethyl Corp. v. EPA*, 541 F.2d 1, 24 (D.C. Cir. 1976) (emphasis added). As a result, "[t]he agencies' threshold of proof is reasonably lower than that appropriate in tort law, which 'traditionally makes more particularized inquiries into cause and effect' and requires a plaintiff to prove 'that it is more likely than not that another individual has caused him or her harm.'" *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996) (citation omitted). Thus, courts regularly exclude as methodologically unsound opinions that involve extrapolation from epidemiological studies reporting an elevated risk of disease at very high exposures to conclude that a plaintiff's much lower exposure caused disease.[10] As a result, Dr. Brodkin's references to

[10] *See Betz*, 44 A.3d at 33.

"regulatory standards are not probative of [a] scientific analysis." *Ametek* Order, at 3.

In short, Dr. Brodkin should be precluded from offering an opinion or expert testimony to the effect that Mr. Quirin's alleged exposure to Calidria asbestos participated in causing his mesothelioma merely because it contributed to his aggregate asbestos dose and/or "total risk," or that the exposure was otherwise "substantial" or a "substantial factor." Such testimony amounts to Dr. Brodkin's personal value judgment of what constitutes "cause," defies the standards for admission of expert testimony established in Fed. R. Evid. 702 and *Daubert*, and in any event is plainly incompatible with causation standards that are firmly established in Illinois tort law.

III. CONCLUSION

For the reasons set forth above, Union Carbide respectfully requests that this Court issue an Order granting its motion *in limine* to preclude Dr. Carl Brodkin from offering expert testimony or opinion that Mr. Quirin's alleged exposure to Union Carbide Calidria asbestos participated in causing his mesothelioma or was a "substantial factor" in bringing it about.

Dated: August 1, 2013

Respectfully submitted,
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CERTIFICATE OF SERVICE

I, Richard F. Bulger, an attorney, hereby certify that on August 1, 2013, I caused a true and correct copy of the foregoing DEFENDANT UNION CARBIDE CORPORATION'S MOTION *IN LIMINE* TO PRECLUDE DR. CARL BRODKIN FROM TESTIFYING THAT EXPOSURE TO UNION CARBIDE ASBESTOS WAS A CAUSE IN FACT OR A "SUBSTANTIAL FACTOR" IN CAUSING PLAINTIFF'S MESOTHELIOMA to be filed and served electronically via the court's CM/ECF system.

s/ Richard F. Bulger_____

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EXHIBIT 1

Transcript of the Testimony of

Carl A. Brodkin, MD MPH FACOEM
December 17, 2012

Quirin v. Alcatel-Lucent USA, et al.
No. 12 L 005290

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IN THE CIRCUIT COURT OF COOK COUNTY, ILLINOIS

COUNTY DEPARTMENT, LAW DIVISION

RONALD J. QUIRIN and MARILYN QUIRIN, Plaintiffs, vs. ALCATEL-LUCENT USA, INC., et al., Defendants.	No. 12 L 005290

DEPOSITION OF CARL A. BRODKIN, MD MPH FACOEM

December 17, 2012

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EXAMINATION INDEX

EXHIBIT INDEX

EXHIBIT INDEX (Continued.)

EXHIBIT NO.	DESCRIPTION	PAGE NO.
Exhibit No. 6	Folder containing billing documents.	18
Exhibit No. 7	Folder containing Kent Micronite documents.	18
Exhibit No. 8	Folder containing Western Electric documents.	19
Exhibit No. 9	Folder containing Lucent Technologies documents.	19
Exhibit No. 10	Folder containing Motorola documents.	20
Exhibit No. 11	Folder containing Georgia-Pacific documents.	20
Exhibit No. 12	Folder containing United States Gypsum documents.	20
Exhibit No. 13	Folder containing Crane Company documents.	21
Exhibit No. 14	Folder containing Ingersoll-Rand documents.	21
Exhibit No. 15	2-page document containing cover letters to Waters & Kraus dated 12/14/12 and 12/15/12.	22
Exhibit No. 16	3-page document containing cover letters to Dr. Brodkin from Waters & Kraus dated 10/5/12, 11/8/12 and 12/10/12.	22
Exhibit No. 17	Curriculum Vitae of Dr. Brodkin.	22
Exhibit No. 18	64-page document containing handwritten notes.	88

EXHIBIT INDEX (Continued.)

EXHIBIT NO.	DESCRIPTION	PAGE NO.
Exhibit No. 19	14-page article, Environmental Exposure to Asbestos and the Exposure-Response Relationship with Mesothelioma.	80
Exhibit No. 20	9-page article, Blood Superoxide Dismutase and Plasma Malondialdehyde Among Workers Exposed to Asbestos.	83
Exhibit No. 21	7-page article, Mesothelioma in Egypt.	85
Exhibit No. 22	14-page article, Mesothelioma in Drywall Finishing Workers.	89
Exhibit No. 23	3-page article, Discussion on "Mesothelioma in Drywall Finishing Workers."	90
Exhibit No. 24	9-page article, The CARET Asbestos-Exposed Cohort: Baseline Characteristics and Comparison to Other Asbestos-Exposed Cohorts.	93
Exhibit No. 25	11-page article, "Re-Creation of Historical Chrysotile-Containing Joint Compounds.	107
Exhibit No. 26	20-page article, A Biopersistence Study Following Exposure to Chrysotile Asbestos Alone or in Combination with Fine Particles.	118
Exhibit No. 27	Folder marked Charles Burns.	201
Exhibit No. 27A	Folder from Charles Burns marked Kent Micronite/P. Lorillard documents.	201

EXHIBIT INDEX (Continued.)

EXHIBIT NO.	DESCRIPTION	PAGE NO.
Exhibit No. 27B	Folder from Charles Burns marked Kent Micronite/Hollingsworth & Vose.	201
Exhibit No. 27C	Folder from Charles Burns marked Kent Micronite Discovery.	201
Exhibit No. 27D	Folder from Charles Burns marked Dr. Longo testimony 8/18/95, Micronite v. Raybestos.	201
Exhibit No. 27E	Folder from Charles Burns labeled Trial Testimony of Douglas Hallgren, Horowitz versus Raybestos.	201
Exhibit No. 28	Folder labeled William McGuire.	201
Exhibit No. 28A	Folder from William McGuire labeled Lorillard Cases Death Certificates.	201
Exhibit No. 28B	Folder from William McGuire labeled Trial Testimony of Douglas Hallgren, Cox versus Asbestos Corp.	201
Exhibit No. 28C	Folder from William McGuire labeled Owens-Corning Testimony of Kent Micronite/Testimony of Mark Risler, Ph.D.	201

BE IT REMEMBERED that on Monday, December 17, 2012, at 600 University Street, Suite 2300, Seattle, Washington, at 9:43 a.m., before BARBARA CASTROW, CCR, RPR, appeared CARL A. BRODKIN, MD MPH FACOEM, the witness herein;

WHEREUPON, the following proceedings were had, to wit:

<<<<<< >>>>>>

DR. CARL A. BRODKIN, having been first duly sworn by the Certified Court Reporter, testified as follows:

EXAMINATION

BY MR. PFAHL:

Q Good morning, Dr. Brodkin.

A Good morning.

Q My name is Scott Pfahl. I'm here for Georgia-Pacific, and we have met on a number of occasions and recently within the last month or so. And today we're here in the Quirin matter, correct?

A Yes, that's my understanding.

Q I will try not to go over old ground we've already plowed

through to the extent that I can. And I will try to cover things that you've done in this case and to the extent new things that I wanted to talk to you about. And I will lead off, and then others will ask you questions.

First of all, as you always do, you have brought with you a number of records, documents, case materials. And for the record let's go ahead, if we can, and identify those things, and we can mark as exhibits the various parts on the record that you brought with you.

A Sure. And to hopefully simplify things, on the -- on Page 3 of a subgroup of notes entitled Clinical Summary and Materials Reviewed, I have cataloged the materials I reviewed. And they include a direct examination of Mr. Quirin's x-ray and imaging studies, and I do have a page of notes from my examination of those, a direct telephonic interview with Mr. Quirin, and I have my notes from that interview, review of medical records, and my notes should be another subsection of the documents you received from my office, pathologic reports, billing documents, deposition testimony, including three volumes of Mr. Quirin's deposition, and then two coworkers, Daniel Di Fazzio and Jack Williamson, Mr. Quirin's naval personnel documents, a work history sheet and various discovery documents.

I have brought in notes from those discovery documents, but to the extent that I pulled any of those discovery documents and looked at them in this case, I did bring them in with me today.

Q With respect to the materials you brought with you, you have what is a case specific file, correct?

A I do, yes.

Q And let's set aside your notes, which for many are in the nature of a report, for the time being, and let's identify the other materials you brought with you --

A Sure.

Q -- today.

A Yes, the case specific file for Mr. Quirin is in an expandable Redweld, six-inch Redweld, and it basically includes manila folders from the materials that I spoke to you about earlier. I can just read through them, if it would be useful?

Q Sure.

A The first one is entitled Work History Sheet and Naval Documents. The second one is Deposition Testimony of Ronald Quirin. The next is Coworker Declarations and Depositions. That would refer to Mr. Di Fazzio and Mr. Williamson. Pathology is the next folder. Chronologic Medical Records is the next folder, and then Comprehensive Medical Records is the next folder.

Billing Documents is the next folder.

And then there's some discovery documents that I received in this case. The first one is Kent Micronite, Including MAS Testing from July of 2012, and MVA Testing by Millette from September of 2010.

And then the next manila folder is Western Electric. The next is Lucent Technologies. The next is Motorola. The next is Georgia-Pacific. The next is United States Gypsum, the next is Crane Co. And the next is Ingersoll-Rand.

And then I have another two sets of expandable Redwelds in a box adjacent to me that includes materials in broad overview relevant to Kent micronite filter exposure.

Q If you wouldn't mind handing me your Redweld?

A Sure.

Q (Peruses documents.) And the first manila folder was Work History Sheet and Naval Documents for Mr. Quirin -- Quirin or Quirin?

A I say Quirin.

Q He didn't object --

A He didn't object when I said it.

Q For Mr. Quirin. These -- this is just the standard work history sheet that we all have through production. I would be inclined not to mark it unless somebody else

would like me to.

And then the other item in the Work History folder was the naval documents. And I believe this was produced also in discovery. I will make a note here for the record that there are some Post-its on here.

And, Doctor, you put the Post-its on the naval records?

A Correct. All Post-its on the documents are mine.

Q There's a Post-it which just indicates the ship, which is the Tolovana?

A Yes.

Q And then also a date, and that's when he was first assigned to the Tolovana as far as you understand?

A Correct.

Q And there's another Post-it 9/4/53, and I'm not sure what is written on this one. If you could just identify that for us?

A (Peruses documents.) That is the assignment to Great Lakes. That was the naval station for training.

Q Okay. That's the training date?

A Yes, or probably enlistment date, close to it.

Q And finally mechanic rating is indicated?

A Yes.

Q And I will set that aside. If anybody would like it to be marked, they may ask for it to be marked.

Page 14

Now, there is a folder that you identified as Mr. Quirin's deposition testimony, and here there are a number of markings and Post-its that you have put on the testimony that you reviewed; is that correct?
A That's correct.
Q And I take it that you did that as a way to help you find things should you need to look at the testimony again?
A Yes. They are identifying Post-its, and they refer to markings on the page. They also are the basis for my coming back and taking notes.
So the 23 pages of Occupational and Environmental History certainly would reflect those Post-its, but they do identify specific pages.
Q All right. There are portions that are circled, you have underlining, stars by some of the testimony, correct?
A Yes.
Q And since that -- this contains those type of personal markings, then we'll go ahead and mark as Exhibit No. 1, the Quirin testimony which has your notations on them.
(Exhibit No. 1 marked for identification.)

Q (By Mr. Pfahl) The next folder is the coworker declaration and deposition testimony. Similarly, you have made marginalia markings on these items, correct?

Page 15

A That's true.
Q And there are also Post-its there with identifying information?
A Yes, a similar process.
Q I will mark that as Exhibit No. 2.
(Exhibit No. 2 marked for identification.)

Q (By Mr. Pfahl) There's a third manila folder entitled Pathology that you identified earlier?
A Yes.
Q And it appears that there are similar types of marginalia and Post-it notes on the pathology documents that you have?
A Correct.
Q So that we can memorialize all of this, I will mark that as Exhibit 3.
(Exhibit No. 3 marked for identification.)

Q (By Mr. Pfahl) Another folder that you have in your Redweld is the chronological medical records. These have marginalia and Post-its on them as well, correct?
A Yes.
Q Now, you have a broader set of medical records?

Page 16

A Correct.
Q The chronology -- chronologic medical records that are in this folder, are those a subset of the larger group you were provided?
A Yes.
Q Were these -- the records and the chronology in that folder, are these records that you, yourself, pulled or were they pulled for you?
A I didn't pull them. I received them as a separate group of documents. But I certainly have gone through the comprehensive medical ones, and they are a subsection.
Q I will mark the subset as Exhibit 4.
(Exhibit No. 4 marked for identification.)

Q (By Mr. Pfahl) And then we have the broader set of medical records for Mr. Quirin. And during your review of the broader set, you have put some Post-its and also made some marginalia comments, correct?
A Yes.
Q And I will mark those as Exhibit 5.
(Exhibit No. 5 marked for identification.)

Q (By Mr. Pfahl) The next folder is entitled Billing Documents. And there are Post-its that are attached throughout the record set and also, it appears, some summary Post-its on the front of the first page?
A Yes. The Post-its on the front page are my opinions about the cost of care. So they are substantive summary Post-its. They basically are summations of the individual page billing documents, and they reflect the cost of care indicated in the documents.
There's a second Post-it that indicates some estimated additional costs of care --
Q If you want to take a look? (Produces document.)
A Sure.
Mr. Quirin indicated to me that he received six cycles of chemotherapy. The billing I received only covers the initial two. So I made an estimate for four additional cycles of carboplatin and Alimta.
And then on the third Post-it, I have anticipated some costs, end of life care, palliative care, that typically would be associated with mesothelioma and have provided a total of all those costs as well.
Q For the folks on the phone, what is your total estimated costs for those types of future treatments?
A Sure. The anticipated costs in the future is $70,000, the estimated additional cost of four cycles of chemotherapy is $42,800, the partial existing costs is

Page 17

$291,366, and the total of all of those is $404,166. Those are estimates within a range of uncertainty.

Q I will mark then the billing records with the Post-its and marginalia as Exhibit 6.

(Exhibit No. 6 marked for identification.)

Q (By Mr. Pfahl) There is another folder, manila folder, that has a title of Kent Micronite. And in here, there's an expert report from MAS, as well as an MVA expert report?

A Yes.

Q And both of these contain marginalia and Post-its with notes on them, right?

A That's correct.

Q I will go ahead and mark that as Exhibit 7.

(Exhibit No. 7 marked for identification.)

Q (By Mr. Pfahl) The next folder is titled Western Electric. This contains a two-page document, Memorandum for Record, and you have some Post-its on here, as well as some marginalia, correct?

A Yes.

Q And I will mark that as Exhibit No. 8.

(Exhibit No. 8 marked for identification.)

Q (By Mr. Pfahl) The next folder is Lucent Technologies. In here, you have Defendant Lucent Technologies Responses to Plaintiff's Supplemental Interrogatories and Requests For Production. This was in the Robert M. Taylor versus Bondex matter, which was in Harris County, Texas. And here you have, it looks like, the full interrogatory response; is that correct?

A That's my understanding. I don't know if it's the full interrogatory response, but I was provided this document.

Q It looks like it stops at Page 16 of 16. There's marginalia here, as well as Post-its, correct?

A Yes.

Q And I will go ahead and mark that as Exhibit 9.

(Exhibit No. 9 marked for identification.)

Q (By Mr. Pfahl) The next folder is -- has the title Motorola on it. It is the affidavit of Timothy Bratton. You have some marginalia here, as well as Post-its?

A Yes.

Q And I will mark that as Exhibit No. 10.

///

(Exhibit No. 10 marked for identification.)

Q (By Mr. Pfahl) The next folder is Georgia-Pacific. You have 1995 Response to Interrogatories that were provided to you; is that correct?

A Yes, not in this case.

Q No, right.

A But I certainly considered them, so I brought them in.

Q I will mark that as Exhibit 11.

(Exhibit No. 11 marked for identification.)

Q (By Mr. Pfahl) The next folder is United States Gypsum. These are Defendants' Answers to Interrogatories and Requests For Production In Re: All Asbestos-Related Personal Injury or Death Cases Filed or to be Filed in Dallas County, Texas. And I will mark that as Exhibit 12.

(Exhibit No. 12 marked for identification.)

Q (By Mr. Pfahl) The next folder refers to Crane Company. And these are responses to interrogatories from the case of Steven Haley In Re Bridgeport Asbestos Litigation. I

will mark those as Exhibit 13.

(Exhibit No. 13 marked for identification.)

Q (By Mr. Pfahl) And the final file in the Redweld is Ingersoll-Rand, a manila folder. This has Amended Responses to Plaintiff's General Order 129 Standard Interrogatories. This is In Re Complex Asbestos Litigation Superior Court of California, County of San Francisco, as well as some responses to interrogatories from the Eldon Dickerson and Ruth Dickerson case, Cuyahoga County, Ohio. And I will mark that as Exhibit 14.

(Exhibit No. 14 marked for identification.)

Q (By Mr. Pfahl) And it looks like you have a folder in front of you, a manila folder. Is that just your report?

A Yeah. I mean, this is part of the Redweld as well, but it includes my comprehensive notes, it includes all correspondence, including the cover sheets for the notes, as well as covers for all the materials I received, as well as my CV.

Q Can I go ahead and see that folder?

A Sure. (Produces documents.)

Q I'm going to mark as Exhibit 17 two transmittal cover sheets. One is dated December 14th, 2012, the other is December 15th, 2012, and these refer to the forwarding of your notes to Mr. Henderson of Waters & Kraus, correct?
A Yeah. I may have missed Exhibit 15 and 16. I'm not sure about that.
Q No, you didn't. I did. Thank you.
All right. Exhibit 15 are those two cover sheets.
(Exhibit No. 15 marked for identification.)

Q (By Mr. Pfahl) Exhibit 16 will be Waters & Kraus transmittal letters to you, which identify the various documents, records and materials that were provided, it looks like, beginning first October 5, 2012; November 8, 2012; December 10, 2012.
A Yes.
(Exhibit No. 16 marked for identification.)

Q (By Mr. Pfahl) Exhibit 17 will be a copy of your CV. And, Doctor, is this up to date?
A Yes.
(Exhibit No. 17 marked for identification.)

Q (By Mr. Pfahl) And finally we come to your notes, comprehensive notes. I know that you want those back, but can we mark as 18 the full set of your comprehensive notes?
A Sure. And we can make a high quality copy here today, and then I will take -- I need to take the originals back, but that's fine.
Q Absolutely. But that will be 18.
A Sure.
Q Now, have we identified and/or marked all of the materials that you brought with you in your Quirin file?
A Yes.
Q You mentioned that you also had some additional materials that relate to Kent and micronite filters; is that correct?
A That's correct, from other cases I've evaluated.
Q Would you mind just identifying for the record, and Counsel may ask you to mark those today, but those materials that relate to Kent that you brought with you that you felt were pertinent to your opinions in this case?
A Yes. There are two Redwelds, one for Mr. Burns, which is a case I evaluated previously, as well as a Mr. William McGuire, who I evaluated previously.
I did receive various discovery documents in those

cases that do inform my opinion about Kent micronite. So I certainly perused them in my review of this case and brought them in today.
Q Okay. And have you made reference to those additional materials in your detailed notes?
A Yes. There are notes I've taken from those documents in the past, and I've actually brought those notes with me. These aren't notes I created in this case, but they certainly inform my opinion about this case, so I've included them in my discovery documents that I've reviewed in the notes.
Q And with respect to your Kent notes, do you have those handy?
A Yes. Notes that would relate to Kent are in the clinical -- well, they are in a subgroup of notes called Exposure Related Documents Reviewed and Considered for Kent Micronite. And they would be a subset of notes going from Page 1 to 4. So that would refer to those notes.
There is also a reference list that informs my opinion about exposures related to Kent micronite in the list of articles I've considered, and that would be on the last page of the list. It begins with Dodson and Hammar, Inhalation Toxicology, 2006, at the top and would end in Millette, MVA 2010.

The other notes that relate to Kent micronite really would relate to Mr. Quirin in his occupational and environmental history.
Q Just so I understand, the four pages of notes that are identified as Exposure-Related Documents Reviewed and Considered, those follow your reference and reliance list?
A Yes, they would have been faxed after that.
Q And the specific references in those four pages are based upon a review of the additional Kent materials that you brought with you, but were not in the Quirin file?
A That's correct, yes. Those notes would refer to that.
Q And do you have a set of notes, handwritten notes, that you have over in the files in the box that relate to Kent that you reviewed?
A No. I do have a list of articles that inform my opinion about Kent, and they are in the list of articles that I provided in this case. It's a cumulative list that I keep. I did add the two references I received in this case, Rigler and Longo, MAS 2012 and Millette, MVA 2010 to the list, but it is a cumulative list. I have used it in other cases as well.
Q I think what I'm going to do is find a place for those.
A On a break, maybe we can put them back in there. It might be easier.

Q All right. Doctor, we've now identified and marked everything you brought with you, correct?
A Yes.
Q It appears from some of the cover sheets from Waters & Kraus that you were contacted in October of this year; does that sound about right?
A That's correct, early October.
Q And generally speaking, did you have any specific charge or direction?
A Yes. I had a chance to speak to Mr. Gibbs Henderson in early November of this year after receiving some of the initial documents, and my understanding is that I was being asked to perform a medical evaluation of Ronald Quirin that would include his diagnosis, his occupational and environmental medical history, any asbestos-related effects, including mesothelioma, if there were asbestos-related effects, the scientific and medical evidence of causation, his clinical course and treatment, whether it was reasonable and necessary, the cost of care, whether that was reasonable and necessary, as well as potentially state of the art issues.
Q With respect to medical evaluation, obviously you relied upon medical records that were provided to you?
A Correct.
Q You also had a telephone interview with Mr. Quirin?

A Yes. In my conversations with Mr. Henderson later in December, I did ask for an opportunity to interview Mr. Quirin to understand his medical history. I also had a couple of questions for him for his occupational and environmental history. So I called him on December 12th of this year.
Q And obviously you didn't meet him in person, correct?
A Correct.
Q Is that something that you requested to do or gave some thought?
A I requested it.
Q And is there any reason why you didn't have a chance to meet with him in person?
A Well, if an individual lives close to my clinic, which is over in Kirkland across the lake, I would certainly arrange to evaluate that person in clinic and take the interview in clinic and do a direct examination as well.
For someone in a distant location who is ill and can't travel, that usually is not possible or practical.
So in lieu of that, if possible, I like to do a telephonic interview, at least to understand the clinical course of their illness and perhaps ask any questions I have about the occupational history. But the most important thing is to really understand from the individual the course of their illness.

So that's what I did. I do rely on the medical records for the objective examinations, which I've done in Mr. Quirin's case.
Q Did you speak to any of Mr. Quirin's treating physicians about their care and treatment of Mr. Quirin?
A No.
Q Now, you indicated that you wanted to speak with Mr. Quirin because you wanted to follow up on a couple of items. One was medical history?
A Yes.
Q And was he able to clarify that to your satisfaction?
A Well, he certainly updated his medical history, which was useful, and certainly we went over his current symptoms and how he was being followed by Dr. Alikhan, his oncologist, and what the plans were for follow-up. So, yes, I certainly updated it.
He also confirmed his past medical history and history of present illness. So certainly I correlated that with his deposition, as well as the medical records.
Q And did you find that your discussion with Mr. Quirin about his past history was consistent with the medical records that you saw?
A Well, I would say overall it was consistent in terms of the types of medical conditions he had been treated for, some borderline or mild hypertension, borderline

diabetes, some arthritis in his knees. Those certainly correlated with the medical records.
He indicated to me and confirmed that he was a former smoker. That certainly correlated with his deposition and medical records. As is common, there certainly is variation in the pack years reported in the medical records and what he reported to me.
But I would say the overall timeframe of smoking is quite consistent. He reported a use of half a pack per day. In the medical records, it was heavier, a pack a day or perhaps more. So some of the providers had a higher cumulative pack year than he was reporting. But his reporting was very consistent with the deposition and the interview.
Q And did you have any -- or glean any additional information from Mr. Quirin in the interview about his occupational and environmental history or potential exposure?
A I did go over a couple of points. Mr. Quirin indicated to me in the interview that, you know, while he had a very good memory of the types of things he did, his memory as to specific dates were somewhat limited.
But he did go over some of his activities. We talked about the bystander exposure to drywallers. I wanted to get a feel for how often he was exposed to

drywallers. I would say his answers were not more specific than the deposition. He indicated it was quite often.

I did clarify the type of sanding he was near. It was pole sanding. That was not asked of him in the deposition. And I tried to get a feel for dry-mix compound versus premixed, and I would say he was not more specific than he was in the deposition. He occasionally saw premix, most of it was dry mix.

The other area we talked about was cable vaults. I wanted to get a little bit more understanding of what he did with that. And I think his description was consistent with his deposition testimony. Again, he wasn't specific as to, you know, the exact number of times he did it or the specific years, but he did indicate it was something he did frequently, particularly in the late 1960s when he was on a cable crew, and he did it at other times as well.

And he described the cable vaults to me as being associated usually with the larger telephone systems with switchboards and key sets where cables would enter these holes, and they would have to be filled.

And then I went over his subsequent work history after he was a telephone installer and supervisor because that really wasn't covered much in the deposition. He

did indicate he worked for about four years in the late 80s as a sheriff's deputy, transported prisoners to court in the Chicago area.

And then when he relocated to North Carolina in the 1990s, he worked as a night supervisor at a grocery store doing some stocking and cashier work.

And then between about 1999 and around 2006, he was a supervisor at Wal-Mart part time.

And we talked about his environmental history. He did not do any vehicle mechanical work, any brake work. That was not reviewed in the deposition. He didn't do any home remodel projects of significance. He did elude to one project where he assisted his son, I think, on a wall using drywall joint compound that was covered in the deposition.

We went over his cigarette smoking history. I've indicated that before. We talked about the start of his cigarette smoking, his experience in the Navy, how he smoked and the brands he smoked. We went over his use of Kent micronites in the Navy. That's all indicated on Page 2 of my interview notes.

But I would say really it was quite consistent with what he indicated in the deposition.

Q Thank you.

MR. PFAHL: Off the record for a

minute.

(Discussion off the record.)

Q (By Mr. Pfahl) All right. Dr. Brodkin, with respect to your evaluation of Mr. Quirin and his various diagnoses, did you come to any conclusions?

A Yes, and I do have a section in my notes that would have been faxed in the second group that summarizes my diagnosis and assessment based on all the evidence. That's a group of notes, subset of group of notes, from Page 1 to 8. It should be in the second group that you received.

The first line would be diagnosis malignant pleural mesothelioma, left side. And basically --

Q Let me see if I can catch up with you.

A Sure.

Q (Peruses documents.)

A It would probably be after Clinical Summary and Materials Reviewed in the second set of notes that you got. This is what it looks like. (Indicating.)

Q (Peruses documents.)

A That's the first set.

Q Oh, okay. All right. Thank you.

And I'm sorry I interrupted to find out where we were, but this is the third page of materials that we

received this morning, which was the second set of documents that you -- or notes that you sent to Waters & Kraus, correct?

A Yes. It is a second group.

Q And I'm sorry. I interrupted you. Please go ahead.

A No problem. Basically, it does provide my diagnosis and the bases for my diagnoses in the subgroup of notes. But I did diagnose malignant pleural mesothelioma on the left side. It's my opinion that it is causally related to various occupational exposures that he encountered in the course of his naval service, as well as subsequent career as a telephone installer and supervisor with additional environmental exposure to the Kent micronite cigarettes between mid 1954 to mid 1956 to a high degree of medical certainty.

And then the bases for the exposure are really a consideration of the nature and extent of asbestos exposure, and I do provide a detailed discussion of that in the notes.

Then the clinical and epidemiologic evidence for asbestos-related mesothelioma, thirdly, a consideration of latency and, fourth, a consideration of differential diagnosis assessing whether there are any other risk factors for mesothelioma or clinical deterioration.

Q With respect to your diagnosis of mesothelioma, you

indicated there was a left-sided pleural, correct?
A Correct.
Q Did you find evidence of any other asbestos-related diseases for Mr. Quirin?
A No, not in his medical records or in my review of his radiographic and imaging studies. Certainly the malignant mesothelioma is a signal marker for asbestos exposure, but in terms of nonmalignant markers, I didn't see any evidence of plaques on the contralateral right side or evidence for asbestosis.
He didn't have a high resolution CT scan, but certainly there was no indication in any of the treating physicians or in my review that he would have had a preexisting asbestosis.
Q You indicated -- well, in your notes I believe you stated that you felt that he met the Helsinki criteria for attribution of his disease to asbestos exposure?
A Yes. Based on the nature and extent of exposure, which is a significant occupational and environmental history of exposure. And then based on the clinical evidence of pathologically proven mesothelioma, as well as a consideration of the epidemiologic studies indicating risk for mesothelioma in similar workers and a consideration for the differential diagnosis, those are all considerations that would meet the Helsinki criteria

for an asbestos-related malignant mesothelioma.
Q You indicated that his course of disease was important to you in your determination of the diagnosis and potential cause, correct?
A Well, the course of the disease is important clinically in terms of the diagnosis. I would say an asbestos-related mesothelioma wouldn't have a course distinct from a non-asbestos-related mesothelioma. But the course of the illness is important, as well as the pathology in terms of establishing the diagnosis clinically. So I do consider that.
Q All right. And with respect to diagnosis, itself, whether or not a mesothelioma is caused by exposure to asbestos or ionizing radiation, that wouldn't matter to the actual diagnosis of the disease, correct?
A Not in terms of the pathologic diagnosis. Obviously those risk factors would be important in terms of causation and in terms of considering them.
Q With respect to treatment, the actual cause of a mesothelioma, be it asbestos or some other cause, would be irrelevant for determining the treatment course, correct?
A That's right. The treatment for a malignant mesothelioma would be the same regardless of the cause. It would be based on staging, clinical issues, efficacy of treatment.

Q There are some references in the second set of notes that were provided, and they were forwarded to us this morning, medical treatment reasonable and necessary is a reference. You are being asked to give an opinion as to the reasonable and necessary nature of his treatment; is that right?
A Correct.
Q What are your opinions with respect to that?
A My opinion is that the treatment Mr. Quirin received was reasonable and necessary. Basically, the consideration following his pathologic diagnosis in December of 2011 with a biopsy that was done by thoracoscopy was whether he would benefit from surgical therapy.
A PET scan was done in January of 2012 that indicated the disease was fairly circumscribed to the left hemithorax. There wasn't evidence of mediastinal lymphatic spread.
So I think given that, the consideration for a radical pleurectomy decortication procedure was reasonable, and that was done at the University of Chicago in February of 2012.
Unfortunately, intraoperatively they did find more advanced disease. Two of his five mediastinal lymph nodes were positive for metastases. His tumor had extended into the peritoneum transdiaphragmatically and

had extended into the pericardium as well.
So he was a Stage IV advanced mesothelioma. That wasn't known prior to the surgery, but it certainly was an effective debulking procedure, and I agree with the adjuvant chemotherapy based on the extent of disease. And certainly carboplatin in a 70-plus-year-old man would be reasonable, along with Alimta.
Q There's a reference in your notes to life expectancy?
A Yes. Certainly a consideration of his life expectancy as a 77-year-old male before his diagnosis of mesothelioma. I've also provided some estimates in my clinical discussion based on his current diagnosis of mesothelioma.
Q With respect to your estimates of Mr. Quirin's life expectancy now, what are they?
A In terms of his advanced stage mesothelioma, he has a predominantly epithelial mesothelioma. There's a small component that is sarcomatoid. But for epithelial mesothelioma, overall life expectancy is 12 to 18 months from diagnosis. Even with the surgical multimodal therapy, only 38 percent over all individuals survive beyond two years. So he has a very limited guarded prognosis. He's approximately 13 months into his presentation, so he has an extremely guarded prognosis.
Q And his life expectancy in the absence of mesothelioma?

MR. MILOTT: I'm going to object to the expertise that this witness doesn't have to make that response.

THE WITNESS: I considered the actuarial life expectancy of a 77-year-old man and whether Mr. Quirin specifically had any medical conditions that would adversely impact that actuarial life expectancy.

And essentially the mild hypertension was controlled with medication. His diabetes was well controlled, actually borderline controlled with diet. His cholesterol levels were well controlled.

Other than that, you know, with some benign colon polyps and some degenerative arthritis in his knees, he really had no significant limitations, certainly had no clinically significant cardiovascular disease or prior respiratory disease that would adversely impact his life expectancy.

So I think the actual -- the actuarial life expectancy of about nine years for a 77-year-old male would apply to Mr. Quirin.

Q (By Mr. Pfahl) You mentioned that you also have some opinions with respect to the costs of his treatment and future treatment.

Did we talk about those as it relates to the notes

that you identified for us earlier or do you have additional opinions in that regard?

A No, I indicated the estimates based on my review of the billing documents and the medical records and interviewing Mr. Quirin.

Q Now, you mentioned at the beginning that you also had an understanding that you would be asked to give some opinions in general about state of the art; is that right?

A Yes.

Q And to this point, what have you been asked to do as it relates generally to state of the art?

A Well, my opinions regarding medical state of the art are really the evolution of knowledge about diseases caused by asbestos over time, and that would date from the early 1900s to the current time as that knowledge evolved.

And, similarly, it would include the communication of that knowledge in terms of recommendations. Many of the articles that I've indicated in my reference review list refer to state-of-the-art articles that inform my opinion about that evolution of knowledge.

Q With respect to the evolution of knowledge, do you hold any opinions as to the knowledge level for medical causation as it relates to asbestos-containing joint compound products?

A I have indicated a number of articles that inform my opinion about that. They would be on the fifth page of the reference list. It would start with Dement, Journal of American Society of Testing Materials, and end in Kinkhead and Carpenter at the bottom. It's a page that relates to joint compound generally.

But there are a number of articles that reflect that evolution of knowledge. One I've indicated is Hueper in Occupational Tumors, 1942, where it's discussed that wall board and related cements and plasters are sources of asbestos exposure, and the chapter indicates concern for pulmonary malignancy, lung cancer and asbestosis.

And then Hueper in Annals of New York Academy of Sciences, 1965, addresses mesothelioma in addition to lung cancer and asbestosis. And the Hueper and Hendry articles in that volume discuss joint filler compounds.

So those would be two articles that would specifically address medical concerns. I would say Soule in the Gypsum Association, 1973, discusses exposure levels with mixing and sanding and does indicate that minimal nonoccupational exposure is sufficient to cause mesothelioma. So that concern is expressed.

And then, of course, you know, based on studies in the 1970s, The Consumer Product Safety Commission's paper in the Federal Registry, 1977, regarding risks of various

asbestos-related diseases, including mesothelioma associated with joint compound.

Q Doctor, we've spoken before about asbestos fiber type potency?

A Yes.

Q And I don't want to rehash all that, but generally speaking you are of the belief that amphibole asbestos fibers are more carcinogenic on a fiber-per-fiber basis than are chrysotile fibers, correct?

A My opinion based on my review of the medical evidence is that amphibole fibers are several times more potent than chrysotile in causing mesothelioma. That wouldn't apply for lung cancer or asbestosis, but for the disease end point mesothelioma, yes, in the range of about threefold increased potency.

But certainly all the fiber types including chrysotile are potent carcinogens.

Q With your -- excuse me.

With respect to your opinions about chrysotile and potency for cancer, does it matter to you in your opinion about chrysotile and its ability to cause various cancers, including mesothelioma, as to the location of where the chrysotile was mined?

A Well, the location where chrysotile was mined certainly is reflected in some of the epidemiologic studies, and

those areas certainly inform my opinion that chrysotile is potent in causing mesothelioma because there are certain areas where relatively pure chrysotile was either mined or the end products -- or manufactured and the end products were conducted on relatively pure chrysotile.

So certainly areas of the world where that occurred, and I have reviewed a number of them on the reference reliance list, but, you know, they would include certainly the Quebec experience where in the Township of Asbestos there's relatively pure chrysotile. In the South Carolina and North Carolina textile industry, there was relatively pure chrysotile. In the mining and milling areas of Balangero, Italy, there are high incidences of mesothelioma.

In Chungking, China, Yano certainly has documented high risk for mesothelioma in workers exposed to relatively pure chrysotile. The same would be true for Zimbabwe miners and millers. So -- and there are other industries as well. I mean, if you look at the New Orleans asbestos cement industry, there were areas of plants where chrysotile was used, and they certainly experienced mesothelioma, workers in those plants.

So in looking at those areas, it certainly informs my opinion that chrysotile is a potent cause of mesothelioma. I guess, you know, Madkour would be

known human carcinogen. The fiber types are not distinguished in terms of their ability to cause cancer.

So clinically it's really not a consideration. I mean, even if something is a mixed fiber type, it's really the dose that's most important. But I certainly would consider, you know, qualitatively if there's exposure to an amphibole versus chrysotile, I mean, there is some relative potency difference.

Now, in terms of contaminants, no, that's really not something I spend a lot of time with.

Q Oh, okay. That's what I wanted to ask you though. The relative level of a contaminant in chrysotile like tremolite, for example, it sounded from your answer like that would be at a level where it would not affect your opinion on the potency of chrysotile to cause mesothelioma; is that correct?

A Well, I think when one is dealing with contaminants, certainly the main clinical effect is going to be the major fiber type present.

So it's the dose of asbestos that's important to my consideration in terms of, you know, the intensity and the duration qualitatively in the occupational and environmental history. That's what I consider.

Certainly the presence of amphibole as a contaminant may increase the risk. I mean, it presents an amphibole

another, a plant in Egypt that, you know, over time there were various fiber types used, but certainly he reports chrysotile asbestos cement pipe and reports numerous cases of mesothelioma.

Q Now, in your opinion can anthophyllite cause mesothelioma in humans?

A Yes. That's not a commercially used fiber type, but it is an amphibole, so yes.

Q And so, yes, anthophyllite can cause mesothelioma in humans in your opinion?

A It would have the properties of an amphibole, so yes.

Q Now, you mentioned a number of different either mines or areas where chrysotile was used and mesotheliomas were reported. My question -- let me try to re-ask this and be more specific for you.

In your understanding of chrysotile and its relation to mesothelioma in humans, does your opinion vary at all as to the potency for chrysotile to cause mesothelioma in humans depending on whether or not that chrysotile was mined in the Carey Canadian mine in Canada or in a mine in South Africa?

A No. As a physician in occupational and environmental medicine, it doesn't. In my opinion, I certainly would cite Lancet, 2009, in terms of the International Agency For Research and Cancers's position on asbestos as a

fiber and some potential for a mixed fiber exposure to the extent there is contamination. But in certainly a trace contaminant, it's going to be the major dose response of chrysotile that's the risk for the disease outcome, not the contaminant.

I mean, the contaminant is something I would consider that, you know, maybe there is some degree of mixed fiber, but it's going to be a much smaller effect than the dose of chrysotile in my opinion.

Q Okay. Now, if we have a product that has chrysotile in it as one of the constituents, and under that circumstance, so you have an end user using a product that has chrysotile in it, does your opinion about an exposure to the chrysotile in a product where it is respirable, does your opinion vary at all with respect to which mine the chrysotile came from that ultimately went into use in the product?

A In broad overview, no. I mean, if I had specific information and a product description or material safety data sheet, I would consider it to inform my opinion about the fiber types. I wouldn't discard it.

But it is the dose. The intensity and duration is the major driver of risk, not the contamination. So it would be a minimal consideration.

Q Right.

So it would not be a significant consideration in your opinion as to the potential for an asbestos-containing product to cause mesothelioma where the asbestos is chrysotile if the chrysotile came from -- or was Calidria chrysotile as opposed to Johns-Manville chrysotile from Canada?

A No, it wouldn't. If I were designing an experiment to look -- or looking up an experiment to inform my opinion about the effects of pure chrysotile, I certainly would look for something like Calidria, which is, you know, a NIOSH model pure chrysotile to look at that effect.

But in terms of clinically looking at disease risk, no, I wouldn't distinguish between the two.

Q Have you reviewed any studies that analyzed bulk ore samples from the Carey Canadian mine area of Canada?

A I've read medical articles regarding Carey Canadian mines. It's my understanding that those have relatively pure chrysotile. But I'm not a mineralogist. I mean, I don't spend a lot of time looking at mineralogic studies of these various mines. To the extent they are reported in the medical literature, you know, I have some awareness of them.

Q And just to clarify, even if there were studies that analyzed ore samples from Carey Canadian mines, the mine area, and found no asbestiform amphiboles in it, that

would not change your opinion that chrysotile from the Carey Canadian mine is capable of causing mesothelioma in humans, correct?

A That's true. And it is my understanding that asbestos mined from the Carey mines is relatively pure chrysotile.

Q Under your diagnosis and assessment you have on Page 6, you have a reference for epidemiologic evidence for asbestos-related meso?

A Yes.

Q It says, "Epidemiologic studies of vessel-based workers," and I will just stop there.

Are you aware of epidemiologic studies that find that merchant seamen who worked aboard World War II era military or naval vessels are at an increased risk for developing mesothelioma?

A Yes. And, basically, this paragraph on Page 6 is making reference to my reference reliance list of articles. And on Page 3, the article -- well, the articles beginning with Polakoff and Horn, 1979, and ending in IARC, Lancet, 2009, review much of that literature. But, yes, certainly seamen working aboard older vessels in historically relevant periods may have experienced asbestos exposure, and there are a number of studies here that indicate increased risk for mesothelioma.

Q All right. Your notes are clear, which is much

appreciated. But can you in a thumbnail identify for us those ways that you believe Mr. Quirin was exposed to asbestos during his time aboard the Tolovana?

A Yes. That certainly would be reviewed in the occupational and environmental history subsection of my notes from the first day -- or the first group of notes that I forwarded. And I discuss the 1953 to 1957 fireman and machinist mate part of Mr. Quirin's career. And this really would relate to the May 1954 to 1957 period aboard the USS Tolovana.

And it would go through Page 6. That experience aboard the USS Tolovana and the sources of asbestos exposure and activities are discussed in that section of the notes.

But in broad overview, Mr. Quirin was an auxiliary machinist's mate and fireman. He was responsible for equipment, which was mainly pumps and valves in the non engine room, non fire room areas of the vessel, which were three pump rooms forward, mid ship and aft, as well as the deck winches.

And his job was to maintain and repair them. And in the process of doing that, he described regularly working with gasket and packing material that during that timeframe in the hot steam-powered applications that he was working with would have to a high degree of medical

certainty represented asbestos-containing materials.

In addition, to access the equipment he encountered insulation, including asbestos blankets and insulation cement that he mixed and removed, as well as removal of some pipe covering that also would have represented asbestos-containing materials.

So those would have been regular sources of exposure when he was working with the pumps and valves in the pump rooms and the deck winches, as well as some of the other areas, the laundry area and the well deck.

Q He indicated that from time to time and apart from the work he did with gaskets and flanges and pumps, that he would have to repair pipe insulation, steam pipe insulation, correct?

A Yes.

Q He described that as being half rounds?

A Yes.

Q And he would have to cut those from time to time so that they fit into an area of damaged insulation?

A Yes, he did that.

Q And have you reviewed any naval product specification records that would indicate what type of piping insulation was called for for hot or steam pipe applications?

A Well, I haven't reviewed any specifications for the

Tolovana, and I don't usually review naval specifications because I'm not a naval vessel expert. So I don't review those type of documents typically.

But certainly from the medical literature, it's my understanding during those timeframes that they likely, aboard a naval vessel, World War II era, would have represented some mixed fiber exposure, likely a combination of chrysotile and amosite.

Q And you are familiar with 85 mag products?

A Yes.

Q And that was 85 magnesium and 15 percent asbestos products?

A Yes, 85 magnesia and 15 percent either chrysotile or amosite.

Q Okay. Based on his description of the steam pipes and the locations where he was replacing insulation, would you expect that those applications would have been -- or would have called for amosite asbestos?

A Well, it's likely they included mixed fiber. I mean, I can't speak to the specific breakdown of those components. I mean, overall, you know, both of those fiber types were used aboard naval vessels, and it's my opinion they would have been used in insulation material aboard that vessel. But I can't really speak to the percentages of them.

my opinion about increased risk of mesothelioma among machinists and mechanics.

Q And under what circumstances other than as Mr. Quirin has described himself would mechanics and machinists come into contact with asbestos-containing material?

A Well, not unlike Mr. Quirin, machinists are tasked with maintenance and repair of equipment that in historical periods in hot applications typically involved asbestos-containing materials, and not unlike Mr. Quirin would include gaskets, packing and insulation.

Q Oh, and I meant to ask you in your opinion what was the makeup of the cement product that he mixed and applied over top of the pipe insulating material?

A The insulating cement mud would have been likely short fiber chrysotile. But, again, I can't be specific. I mean, I don't have the specific material descriptions for those. Is it possible there could be mixed fiber? Yes. But I would say in most of those applications, it would be short fiber chrysotile.

Q Based upon your knowledge of the industry, would you expect asbestos, amphibole asbestos, to be used where the insulating material, the cement material, would be exposed to the seawater?

A That I really can't speak to. That would be a technical question that I just don't know the answer to.

I mean, chrysotile was overall by far the most widely used insulation material in North America. But in a naval vessel specification, it certainly would have included amosite in the insulation.

Q And based upon your review of the testimony and information that you have, you assume that Mr. Quirin was exposed to -- or had a mixed fiber exposure during his time on board on the Tolovana?

A It's my assessment based on my consideration of Mr. Quirin's occupational and environmental history that he did experience a mixed fiber exposure during his naval vessel activities as I've described them.

Q Thank you.

Now, in your epidemiologic section of the diagnosis and assessment, we were looking at vessel-based workers. There's also a reference there to mechanic and machinists?

A Yes.

Q And do you have reference to epidemiologic studies which would show an increase in the instance for mesothelioma for mechanics and machinists?

A Yes. On the fourth page of my reference list, beginning with Teschke, Canadian Journal of Public Health, 1997, and ending in Gennaro, Scandinavian Journal of Work and Environmental Health, 1994, I review studies that inform

Q Well, the next group is construction workers utilizing drywall/plastering materials?

A Yes.

Q And we've talked at some length about those type of studies, so I'm going to move on for now.

A Sure.

Q Then you have chrysotile-exposed workers --

A Yes.

Q -- after that as another group?

Can you identify for me who the chrysotile-exposed workers are?

A Yes. We talked about them to some degree in my response to a previous question about areas in the world where there was relatively pure chrysotile. But those articles are reviewed on the seventh and eighth page of my reliance list. It includes a number of those articles that I spoke to and certainly informs my opinion that workers working with chrysotile in general are at increased risk for mesothelioma.

Q Then next you have crocidolite-exposed individuals?

A Yes.

Q As a sub -- as a group.

And do you have epidemiological studies that refer to crocidolite workers or exposed workers?

A Yes. In that same group of notes, certainly I have a

number of articles that look at crocidolite as a specific fiber, and certainly that would start with Wagner in British Journal of Industrial Medicine, 1960, and I have cited that experience in the North West Cape of South Africa.

And then on the final page of the reference list, there are a number of articles that look at crocidolite-exposed workers. Some of them are associated with the Kent micronite filter, others are associated with chrysotile gas mask workers, workers using chrysotile to fabricate gas masks, both in Britain and Canada. So they certainly inform my opinion about --

Q Chrysotile gas masks or crocidolite?

A Well, chrysotile and crocidolite and comparing them.

Q In the supplemental -- what I will say supplemental Kent materials that you have with you, do you have any documents that relate to disease associated with manufacturing of Kent filters?

A On the final page of my notes, there certainly are articles that inform my opinion about that. It certainly would include the Talcott, New England Journal of Medicine, 1999, article. Let me see if I can find it here. (Peruses documents.) Yeah, it's about a third of the way down on the last page.

But it looked at the Massachusetts cohort of

potent cause of mesothelioma.

And I guess just to be responsive to your question in terms of ancillary materials, I have in my notes indicated industrial hygiene investigations at the Massachusetts plant that manufactured the crocidolite filters for the Kent micronite, and certainly they cite health concerns as well. That would be in the group of notes labeled Exposure Related Documents for Kent Micronite.

And on Page 3 of that subgroup, industrial hygiene evaluation of H&V Specialties Company, Hollingsworth & Vose Specialties Company, it's an evaluation of the West Groton, Massachusetts, plant. And they certainly talk about significant asbestos exposure levels and concern for illness with a recommendation for medical exams and chest x-rays among exposed workers.

MR. PFAHL: We've been going for a little while. Do you want to take a break, five minutes?

THE WITNESS: Sure. Five minutes.

MR. KRUKA: Okay.

(Recess from 11:07 to 11:16.)

Q (By Mr. Pfahl) Dr. Brodkin, you have a reference on Page 6 of your Occupational and Environmental History to work practice and hygiene. And, again, this relates to the

cigarette filter manufacturers that were exposed to crocidolite, and of the 33 workers, 5 developed mesothelioma. So 15 percent incidence, that's among the highest rates in the world for development of mesothelioma.

I guess the other area, and I haven't specifically written down these -- these articles, but certainly asbestos cement manufacturing has been studied extensively and would include crocidolite exposure, and there are a number of articles by Magnani that look at the experience with mesothelioma in the Eternit mines in Casale Monferrato, Italy.

And they report in the range of about a six percent mesothelioma rate among that working population, also among the highest in reported working groups.

The other groups that I've indicated that I've talked about are the crocidolite gas mask workers, and Acheson in British Journal of Industrial Medicine, 1982, observed a rate three to four times higher than the chrysotile-exposed gas mask workers.

And McDonald and McDonald in Environmental Research, 1978, found a high rate of mesothelioma, 4.5 percent, among crocidolite gas mask workers.

So those articles, and there are others as well, would certainly inform my opinion that crocidolite is a

timeframe when Mr. Quirin worked aboard the Tolovana and was in the Navy, correct?

A Yes.

Q You indicate that there was no use of respiratory personal protective equipment provided to him, correct?

A That's correct.

Q And he also testified that he had no asbestos-containing material hazard training with the Navy; is that right?

A Correct.

Q With respect to the state-of-the-art studies and articles that you are aware of that relate to asbestos-related disease and causation, for those studies, articles, that were published before 1953, is there any reason why in your opinion that the Navy would have been unaware of those same articles?

A I don't know why the Navy would be unique compared to any other entity in having access to that medical state-of-the-art knowledge. Certainly from the time of Merewether and Price in 1930, methods to control asbestos exposure had been provided and discussed and in follow-up studies and certainly communicated by various entities, including the National Safety Council and the Industrial Hygiene Foundation. I don't know why the Navy wouldn't have access to that as compared to other entities.

Q Let's go ahead and turn over in your Occupational and

Environmental History to the post Navy work and exposure that Mr. Quirin may have had. And the first item was as a deliveryman, and you didn't identify any asbestos-containing materials that Mr. Quirin was exposed to during that short activity, correct?

A Correct.

Q And then that takes us to about 1957 where he becomes a telephone installer, and he joins the International Brotherhood of Electrical Workers at the local in the area where he worked, right?

A Yes, he did.

Q With respect -- or as a thumbnail, what is your understanding as to how Mr. Quirin may have been exposed to asbestos-containing materials initially as a telephone cable installer or telephone installer and repairman, which by my reading was 1957 to about 1966, during that timeframe?

A Yes. That period certainly is discussed in conjunction with the follow-up period when he was a supervisor in 1967 and thereafter on the subset of Occupational and Environmental History notes beginning at Page 7 and going through to Page 20 and certainly detail the sources and activities that would expose Mr. Quirin to asbestos either through direct use of the material or a bystander to use of asbestos-containing material.

In terms of direct use, which you referred to in your question, that would be the installer period, that -- that activity would relate primarily to his work with cable vaults.

And on Page 11 of the Occupational and Environmental History, I discuss the cable hole filler compounds. And the discovery documents indicate that prior to 1974, those were asbestos-containing. And it was Mr. Quirin's practice to mix the dry compound to, basically, secure and waterproof the cable in the holes.

And that was a process that took about five minutes to mix the compound. It was powdery and generated dusty conditions that he felt that he breathed.

And certainly he describes visible dust, and I've cited a number of quotes from his deposition where he indicates that.

And that would have been -- there would have been periods of fairly regular exposure to that in the installer period, which is the 1957 to 1966 period, particularly around 1966 for a six-month period when he worked on a construction gang, worked on numerous sites where he was performing that type of activity.

The other activity that he did with the cable vaults that would have exposed him to asbestos was in fabricating the gaskets between the cover and the hole,

the hole cover gasket.

And the Lucent discovery documents indicate that fire-resistant materials were used in that capacity. So it's likely that the gasketing material would have been asbestos during that period. And he does describe use of a ball peen hammer for that activity that would expose him to asbestos directly. So -- and removal of those gaskets also could result in exposure.

In terms of direct use of materials, the only other potential exposure directly would be the spackle that he was provided to repair walls. That's really not further characterized in the discovery documents, so not all spackles were asbestos-containing.

If what he's describing is a joint compound, in my opinion it likely would have been asbestos containing, but I don't think it's sufficiently characterized where I can say to a reasonable degree of medical certainty that was an asbestos-containing material. So I call that a potential source. If there was asbestos in that, he would have been exposed in the mixing procedure.

Q With respect to the spackle, he identified a ready mix product that came in a small can or that some of his coworkers described that way as well, correct?

A There were ready mix formulations, including a wood filler, various paste, premixed compounds. Those would

not be identified exposure. I mean, even if they were asbestos containing in that form, he didn't describe an activity that would generate airborne fibers.

Q He indicated that to the extent he used spackle to patch some holes, that that was usually done without having to sand; is that right?

A Right. It wasn't his practice to sand.

Q And so in your estimation, a possible exposure for him would be if he was using or mixing up dry joint compound products in order to patch holes; is that right?

A Yeah. I mean, during that period, if he was using a joint compound, it likely would have been asbestos containing. But to me, the term "spackle" is really broader than joint compound. That's why I call it a potential exposure.

Q Then in 1967, Mr. Quirin becomes a supervisor, right?

A Correct.

Q And what is your understanding as to his likely or potential exposures to asbestos moving forward, 1967 and beyond, as a supervisor?

A As a supervisor, his exposures would all have been of a bystander nature. In terms of the cable vault work, he did describe being in proximity to workers that did that work, so this would be bystander, not direct exposure for those materials.

In addition, I discuss bystander exposure really during the whole period that Mr. Quirin was an installer and a supervisor, and there wouldn't have been much difference between his being an installer or a supervisor on the sites because he was basically in proximity to some other trades, primarily drywallers, that used asbestos-containing joint compounds that would have spanned the period of 1957 to '67 as an installer, but also would have spanned a period '67 to about '77 when asbestos was removed from most of those materials.

So that's a 20-year period, whether he was an installer or a supervisor, where there would have been bystander exposure to other trades.

And one that is discussed by Mr. Quirin and his coworkers is a very frequent exposure to workers performing drywall finishing, whether it's mixing, sanding or sweeping. There were also exposures to ceiling tile workers, cutting ceiling tiles, and at various times it's likely that some of those were asbestos containing. They also described some proximity to insulation workers or work in attics that might have exposed them to insulation.

Now, Mr. Di Fazzio describes a lot of that as fiberglass batting, but there was blown-in insulation, which certainly at times could have represented

asbestos-containing material. And certainly Mr. Williamson describes work that Mr. Quirin did around Celotex, which likely was a chrysotile-containing insulation for many of the applications.

So there would have been intermittent exposure in my opinion to asbestos in some ceiling tiles and in some insulation material, you know, at least on an intermittent basis, and then relatively regular exposure to drywall joint compound that would have contained asbestos, and all of that would have been as a bystander.

And he would have continued to have bystander exposure to the cable hole filler compound, at least up to 1974 when there apparently was a transition to non-asbestos material.

Q With respect to joint compound and his bystander exposure to joint compound products or finishing, you said that in your estimation that would have been regular exposure; is that right?

A Yes. Now, Mr. Quirin did not have the resolution of memory of saying, you know, it was a specific number of times per interval, but he certainly describes it as being something that he saw quite often on work sites. Mr. Di Fazzio who worked with him in the '69 to '71 timeframe, this would be when he was a supervisor, he described it variously as one to two times every couple

of weeks.

He also indicated in his deposition that, you know, at times it was more. It could be five days in a row or on some projects it could be three weeks in a row. But it was something that was in my assessment that was regularly encountered on job sites because of what Mr. Di Fazzio did, the pre-wiring activity, basically doing the telephone wiring during the active construction period.

Q All right. Now, with respect to Mr. Quirin's exposure as a bystander to asbestos from joint compound products, was the testimony sufficient enough for you to determine the number of days out of a month that Mr. Quirin was on average exposed to asbestos from joint compound products?

A I would say Mr. Quirin's testimony wasn't to that resolution. And I did ask him those same questions or similar questions when I interviewed him. His description was that it was quite often, but it wasn't the resolution of his memory to say, you know, it was -- it occurred at a specific rate.

Q Okay. You will agree with me that there was testimony from Mr. Quirin, Mr. Di Fazzio and Mr. Wilkinson -- Williamson that the standard protocol -- or not protocol, but the procedure in which for new construction installation of the lines would occur was one where the

electricians would go in and put conduit or piping, especially for commercial projects, and then the phone lines would come in after the electricians, and this was done while the walls were open, correct?

A What you describe I think they consistently indicated would be the ideal sequence of events. They also indicated that that frequently didn't happen, and they would have to essentially adapt to the other workers on the sites, whatever the stage of construction was.

Q Well, they talked about the fact that that was the ideal sequence, it was also the one that happened most frequently, didn't they?

A It certainly did happen, and that was the ideal sequence.

Q And so they said that most of the time, though, it wasn't precisely defined, but most of the time they are not working where there are closed walls and drywallers are finishing the walls while they are trying to fish wire, correct?

A It's certainly not something that happened all the time on work sites. I mean that, I think, was consistent among the coworkers and Mr. Quirin.

Q And then with respect to the work that was going on, Mr. Quirin and the coworkers, they didn't testify that they would just sit around and watch drywallers mixing up joint compound for use on walls, right?

A No. I mean, they were actively working doing their installations.
Q Right. So they were aware of the fact that drywallers would take bags and mix them up, and they called it a mixing bucket or a mixing -- I'm trying to -- a tub?
A Mud tub, yes.
Q Mud tub, right.
But they didn't say they would just sit there next to guys who were pouring in the joint compound and watch them do it, right?
A No, it was something they were aware of and they observed, but it was in the course of their work activities.
Q Right.
The same thing with respect to sanding, they would -- they would describe the drywallers would sand after the mud would dry, they would sand it, correct?
A True.
Q And you said Mr. Quirin indicated in your phone call with him that was usually pole sanding?
A Yes.
Q He didn't indicate how many times he was fishing line through walls in the same room where the drywallers were pole sanding?
A No, but I did ask him the circumstance, and he said they

were often in the same common space or in an adjacent room, not always, but that was something he commonly observed.
Q In your estimation would Mr. Quirin have had the same exposure to asbestos on a dose level as the guys who were actually sanding the joint compound if they were in a room that's 100 feet away from him?
A No. That bystander exposure would be less, and certainly the exposure really relates to the radius from the distance of the activity and the time that's elapsed from the activity.
I have cited a study by Nicholson that informs my opinion, a NIOSH investigation, that was reported in the American Industrial Hygiene Association Conference in 1974. That's on Page 3 of my Diagnosis and Assessment section. And it does provide measurements they took at bystander distances for various specified radiuses and specified times after the drywalling.
I would say on average, those would be less than the direct exposures, but they are certainly significant depending on the radius and time elapsed.
Q And based on Mr. Quirin's testimony or his interview with you, did you have an understanding as to the frequency with which he was in a common room when drywallers were sanding joint compound?

A Not to the resolution of a specific rate or times per week or month. He, again, indicated that he basically worked around the drywallers and often in the same large rooms or adjacent rooms, but his memory was not to the resolution of a specific number of times.
Q Did Mr. Quirin testify as to the number of times that he was in the same room or adjacent to drywallers when they were sweeping up joint compound dust?
A No, it was an activity that he generally described as seeing or observing quite often in the course of his work, but not to the resolution of a specific number of times.
Q Based on Mr. Quirin's testimony, would you be able to calculate a dose on a fiber-per-cc-year basis for Mr. Quirin from his exposure to asbestos from joint compound products?
A Well, it's not something I do as part of my practice, so it's a bit of a moot question. It's not something I would do as an occupational and environmental medicine physician. But I do certainly read fiber cc year dose reconstruction dose calculations. Anyone that was doing that would have to obviously make estimates and assumptions that would be necessarily within a range of uncertainty because there just isn't that specificity of testimony.

Q Right. In terms of -- based upon your review of all the testimony and talking to Mr. Quirin, you didn't see enough specificity with respect to the variable of frequency or the variable of proximity to the activity that would allow a dose reconstruction or estimation, correct?
A Well, I think one could estimate it, but I think -- and typically it's industrial hygienists that do this type of dose reconstruction.
But they would have to make certain assumptions that they would have to state up front within a range of uncertainty. I mean, certainly there's a degree of testimony that indicates that this was a circumstance that occurred on a regular basis. I mean, Mr. Quirin describes it as quite often. I think Mr. Di Fazzio gives a greater resolution of it.
But it is something that happened in my assessment on a regular basis, but it's not to the specificity of a certain number of times.
Q And Mr. Di Fazzio testified about being in a building where joint compound work or drywallers were finishing drywall, but he described being in a different part of the building at the time, right?
In other words, he was aware they were on the third floor, but he was on the fifth floor while pulling wires,

that kind of thing?
A That's a circumstance that could happen as well. I mean, their distances varied depending on the job site. So at times they were in a common room, at times they were in an adjacent room, at times they were more remote, at times it wasn't going on at all.
Q You have references to US Gypsum and Georgia-Pacific?
A Yes, those are the two products that Mr. Quirin indicated he recalled.
Q Okay. And he also admitted that those weren't necessarily the only two joint compound products that were ever used around him, right?
A I think that's fair. It's the ones that he and his coworkers recalled, but not to the exclusion of others they might not have recalled.
Q And do you have an understanding based on Mr. Quirin's testimony as to the general timeframe when he saw -- or he would see one of these products more than the other?
A He did clarify in direct testimony that the US Gypsum -- I think I've indicated this on my notes. (Peruses documents.) On Page 14 of the Occupational and Environmental History, that the US Gypsum would have been in the earlier period of his career when he was an installer. He said he saw that frequently. And then he clarified in his direct testimony that Georgia-Pacific

troubleshoot something at a job, it may demand more of his time, right?
A Yes.
Q But this isn't the situation where he's spending -- typically where he's spending all day at one job site?
A As a supervisor, 1967 and afterwards, I would say not. He was visiting multiple job sites where he would spend variable periods of time in the 5 to 30 minutes up to 3 hours.
Q And some of the times when he would go and visit job sites as a supervisor, he was talking to customers to make sure they were happy with what was going on?
A Yes, or providing estimates.
Q Right. So he wasn't always with his crew who were working on the job site, correct?
A Right. He might be on the job site doing some other activities.
Q You mentioned some insulation exposures. Do you recall Mr. Quirin indicating that there were times when they would have to drag lines through the attic in order to install phones?
A That's true.
Q And back in the 50s and early 60s, there were products called vermiculite insulation that was used in attic insulation --

became the dominant brand that he saw on larger commercial projects when he was a supervisor.
Again, his memory was not to the specificity of giving any definite years or time span, but that was his general recollection.
Q And when Mr. Quirin was a supervisor, he would send out crews to multiple job sites during the day each day, correct?
A Yes.
Q And he would go, and he would visit those crews at least one time a day at the work sites, correct?
A At least once, yes.
Q And he mentioned that he had about an hour in the morning and an hour at the end of the day that he would be in his office doing desk work?
A True.
Q And it was described by Mr. Quirin, as well as his coworkers, that depending on the demands of the particular job and what was going on at the time, he may spend five minutes at a job site?
A There were times when he would spend five minutes, although, the typical span would be a much wider range, 5 to 30 minutes, even up to 3 hours if it was a complex project.
Q Right. And that would depend, if he had to be there to

A It was one --
Q -- blown in?
A It was one type of insulation that could be used. There were multiple ones. It could be fiberglass. It could be cellulose. It could be foam. It could be vermiculite.
Q Now, Mr. Williamson indicated that he was a -- or actually involved with the union and was a safety officer for the union?
A He later became a safety officer later in his career.
Q Yeah. Well, it was during the 70s, right?
A I would have to look at the deposition to the exact point in time, but certainly it was after a decade or so of work at least.
Q He indicated that the union held periodic safety meetings?
A He did, yes.
Q And Mr. Quirin indicated that he attended those safety meetings as well?
A He did indicate he attended some, yes.
Q And Mr. Quirin indicated that he didn't recall asbestos being discussed at any of the safety meetings?
A That's correct.
Q Now, the Selikoff and Mount Sinai group did a lot of industrial hygiene work with insulating unions in the New York/New Jersey area, correct?

A Correct, beginning in the 1960s.
Q Right. And they began publishing by the mid 1960s findings of increase of disease and asbestos-related disease for insulators, correct?
A Yes.
Q And that was something that was communicated to the insulation union workers through their publications, correct?
A That's my general understanding. I can't say I've studied union publications to speak to that with any direct knowledge, but it is my understanding from reading the medical literature that certainly one of Dr. Selikoff's purposes was to communicate findings and concerns for the insulators.
Q And I believe Mr. Williamson testified that he did have an understanding at some point in his role as a safety official with the union that asbestos was something that could be dangerous or harmful? I will put it that way.
A Well, the way Mr. Williamson put it when he was asked was sometime around the mid 1970s, he learned that asbestos was something, I think he put it, that you should keep away from. I don't think it was to the specificity of certain hazards or diseases, but certainly there was some aspect of communication in terms of that.
Q What is the earliest article or study that you are aware

of that was published in the peer-reviewed medical literature that raised a concern about asbestos exposure in the construction trades?
A Well, I mean to some extent in terms of the materials used in the construction trade, that would date to Merewether and Price in part, too, where they would discuss not just their textile findings, but other materials such as insulation materials that could be used in construction.
And certainly in 1932, Merewether and Price talked more about that. But I would say in many of the articles I cite on the first page of my reference reliance list, they talk about state of the art. Many of the reviews of asbestos-containing materials would relate to materials used in the construction trade with that date from 1930 onward.
Q Can you give me specifics in terms of products that would have been used in the construction trade that would have been talked about from the 1930s onward?
A Well, certainly insulation, use of various cements, water pipes, potentially in the mechanical aspects of the heating trades for construction, gaskets and packing as well, as well as rope material. Those would be the general types of materials discussed in these articles over time.

Q And would there have been anything to keep the International Brotherhood of Electrical Workers from learning about those articles?
A While I can't speak to what the IBEW knew and when they knew it because I haven't investigated that. I don't know -- I don't have a reason to think they would have a different access to the medical literature than any other entity.
Q And do you know whether or not the IBEW monitored at all the developments that were going on in the 60s with respect to the asbestos insulation workers?
A I can't speak to that. I haven't investigated it.
Q But it was your understanding that neither Mr. Quirin's employer, nor his union provided any kind of personal protective equipment for him as it would relate to asbestos exposure?
A That's true. During the course of his work as an installer and supervisor, he did not receive asbestos hazard training to his recollection, nor did he use any personal protective equipment in terms of respiratory protection.
Q With respect to Mr. Quirin and his bystander exposure or potential to bystander exposure to asbestos-containing materials, did you stop the exposure timeframe at 1978?
A Basically by 1977, in terms of, well, new insulation, it

would be very unlikely that it would be asbestos containing during that timeframe. And certainly for the drywall joint compound, it unlikely would involve asbestos-containing materials after 1977.
For the ceiling materials, that's probably variable, but as you get past '77, it would be much less frequent.
Q With respect to repair jobs that Mr. Quirin would have supervised, is it possible that his crew could have been exposed to asbestos in place if they had to tear open something, put a new bracket and chip off insulation or some spray material in order to put on a new bracket?
A It's possible there could be intermittent exposures after 1977. I think it would be much less frequent, less likely, but on remodel projects, it's possible.
Q In addition to Mr. Quirin's occupational environmental history -- or not in addition to, but as part of, you also have references to his childhood timeframe?
A Yes, as part of the environmental history.
Q And there you make reference to Mr. Quirin's father who worked for the Chicago and North Western Railroad, and you note that he worked on engines. You didn't know what he did.
Based upon your review of the medical literature, do you have an understanding as to whether or not asbestos materials were used on locomotive engines or along with

locomotive engines?
A Well, prior to 1960 to the extent there were steam locomotives being worked on, there certainly is potential for exposure in terms of insulation material. Again, the history is not well characterized here because Mr. Quirin didn't visit his work site and didn't know what his father did other than work on engines, which is pretty vague in terms of railroad work.
Q Do you assess Mr. Quirin's father's work on engines, railroad engines, as being a potential exposure where he may bring asbestos materials home on his clothing?
A Well, it's one thing I considered. That's why I make a note on Page 21 that in addition to it not being characterized, Mr. Quirin indicated that his father didn't bring his work clothes home.
It doesn't allow me to identify an exposure there, because even if he did work on engines in a way that did expose him to insulation, there's not a cogent root of exposure in terms of an indirect pathway. It's possible, but I can't say to a reasonable degree of medical certainty that it happened based on that information.
Q And then you make reference to surrounding industrial sites.
A Yes, in terms of --
Q What was the reference there about?

A Yeah, he indicated in his deposition that where he parked on Child -- well, near the Child Street Illinois Bell office, that there was a Johns-Manville plant a couple of blocks away. He didn't know what it was doing. He didn't see dust from it. It's really not a characterized site. I've noted it. It's a possible source of area exposure.
But without knowing more, I certainly wouldn't have an opinion about that to a reasonable degree of medical certainty.
Q And didn't he refer to that plant as one that manufactured insulating products?
A Well, he was asked questions about that. He didn't know what it manufactured would have been my assessment of his answer. He just didn't know what it did.
Q And I take it you haven't Googled the Johns-Manville plant in Wheaton, Illinois, to see what they were manufacturing?
A I haven't investigated that plant. I really don't know anything about it.
Q You make reference to one home remodel project where he helped out his son; is that right?
A Yes.
Q And this was one wall that was done?
A Yeah, my interpretation was that it was a fairly limited project.
Q And the remainder of your notes refer to -- not the remainder, but the remainder on Page 22 refer to the Kent cigarettes; is that right?
A Yes, and general cigarette smoking.

(Exhibit No. 19 marked for identification.)

Q (By Mr. Pfahl) Dr. Brodkin, I'm going to hand you an article that I've marked as Exhibit 19. This is one you made a reference to earlier?
A Yeah, the Madkour.
Q This is from The Eastern Mediterranean Health Journal, Volume 15 from 2009. For the record, the title is Environmental Exposure to Asbestos and the Exposure-Response Relationship with Mesothelioma?
A Right.
Q And that's the article you made reference to earlier?
A True.
Q In the abstract, it says, "An epidemiological and environmental study was carried out in Shubra, El-Kheima, K-H-E-I-M-A, city, greater Cairo." Right?
A Yes.
Q And if we turn over to the introduction on Page 26?
A (Complies.)

Q If you look at the right-hand column with me, the first full paragraph there is "Data obtained from." Do you see that?
A Yes.
Q And if we go down to the third sentence there, it begins, "Workers employed"?
A Yes.
Q That sentence states, "Workers employed since 1948 by the Egyptian asbestos company Sigwart at the mills in greater Cairo (El Maasara and Shubra El-Kheima) had an increased risk of mesothelioma, as did former residents of Shubra El-Kheima who were not directly employed in the milling of asbestos."
Do you see where I've read that?
A Yes.
Q And so this gives us a reference point in terms of what they are talking about in terms of what they are talking about for the workers.
And if we look at the next page, 27, under Methods and Location on the left-hand column?
A Right.
Q It says, "This epidemiological and environmental study was carried out in Shubra El-Kheima city, greater Cairo, to evaluate the prevalence of MPM," which would be malignant pleural mesothelioma, right?

A Right.
Q "Shubra El-Kheima is an industrial city at the northern boundary of Cairo, just upwind from downtown Cairo. It has an area of about 30 kilometers. This city was considered the focal point of the highest environmental exposure to ambient asbestos fibers due to the operation of a large asbestos manufacturing plant (the Sigwart Company plant)."
And then it has a figure there which shows its location.
A Right.
Q It says, "The Sigwart plant is an asbestos manufacturing plant using chrysotile asbestos. It was constructed in 1948, and its main products were asbestos cement pipes and reinforced concrete products. The study included six areas in the near vicinity of Sigwart plant."
And I will stop there. All right?
A Yes.
Q And so here's a reference to the plant, its location and the products that it manufactured, correct?
A Right.
Q And in this study, it makes reference to chrysotile asbestos?
A That's correct.
Q All right. And that's why it's of interest to you?

A Well, it's of interest because it certainly is an area-based study and a population-based study of asbestos, and I think it adds additional information about chrysotile asbestos.

(Exhibit No. 20 marked for identification.)

Q (By Mr. Pfahl) I'm going to hand you Exhibit 20. This is a study from Dr. Kamal, K-A-M-A-L, and others from the American Journal of Industrial Medicine, 1992. This is titled Blood Superoxide Dismutase and Plasma Malondialdehyde -- I don't even know what that is. Can you help me with that?
A Malondialdehyde.
Q Thank you -- Among Workers Exposed to Asbestos.
Have you seen this article before?
A I have. I have seen this.
Q You have. Okay. Somebody has shown it to you?
A Well, they've shown it to me, and I have it. So yes.
Q So if we look over on 354, there's a reference in the bottom section Subjects and Methods, right?
A Right.
Q It says, "Subjects were 97 asbestos-exposed workers (mean duration of exposure equals 19.8 plus or minus 8.3 years) randomly selected from Sigwart Company manufacturing

cement asbestos pipes in Shobra El Kheima (a suburb of Cairo). Their job entails transporting the asbestos bales from the store, opening them manually, feeding them together with cement in the mixing machine, supervising the transportation of the mixture to the production machine, transporting the produced pipes to water containers and cutting the pipes into different lengths according to the requested orders. Subjects had no fixed workplace as they interchanged their workplace according to the needed situation. The asbestos used (70 percent white asbestos chrysotile and 30 percent blue asbestos crocidolite) was imported from the United States, Canada and Brazil."
Do you see where I managed to try and read that?
A You've read it correctly, yes.
Q And in this -- here in 1992, they are making reference to the fact that in the manufacture of the cement pipes, they used chrysotile and crocidolite, correct?
A They do indicate that in this section, yes. And -- well, maybe -- I don't know if you have a question about it, but certainly this is, as they say, a large facility, 30 kilometers squared. And certainly I'm relying on Madkour in terms of the description of it being chrysotile asbestos because in large complexes there can be different processes.

And certainly Hughes and Weill looked at the asbestos cement industry in New Orleans that used in areas chrysotile and in areas amphibole. So I'm really relying on Madkour's description of that chrysotile asbestos. I'm aware from Kamal that use of crocidolite occurred in a specific area at the site as well.
Q Well, as it relates to the environmental exposures in the surrounding neighborhoods, the fact that there was crocidolite used would be an important factor in determining what asbestos type may have resulted in the increased incidence of mesothelioma among people who lived near the Sigwart plant but didn't work there, correct?
A I think it's a fair consideration. It does require some knowledge of the site in terms of assessing that, which I don't have. I mean, I can't speak to it. Certainly in citing Madkour, I am relying on his description, but I am aware that other things happened at the broader site.

(Exhibit No. 21 marked for identification.)

Q (By Mr. Pfahl) Let me hand you another article. This one I've marked as Exhibit 21. And it's an article from Eldin, E-L-D-I-N, and others titled Mesothelioma in Egypt.

And first off, does this look familiar to you?
A I don't have it. Whether I've ever been shown it, I can't tell you. I don't -- I mean, I don't have a working memory of it as I sit here, so I would have to read it to speak to it.
Q Sure. Let's look at Page 42, the second page.
A (Complies.)
Q Under the heading Asbestos in Egypt, do you see that?
A Yes.
Q It says, "Asbestos was used since a long time (2000-3000 BCE) as embalmed bodies of Egyptian pharaohs were wrapped in asbestos clothes to offset the ravages of time. Industrialization utilizing asbestos started in Egypt since 1948 by Sigwart Company in El Maasara, South Cairo. Data quoted from the introduction of a training course made by Sigwart Company in 1980 for its employees showed that this industry started by using chrysotile (white asbestos), crocidolite (blue asbestos) and amosite (brown asbestos). Asbestos-using factories steadily increased in number and by the year 2004, 14 asbestos factories were present in Egypt. Data about these factories obtained from the information network of the Central Organization for Industrialization (COFI) is summarized in Table 1."
It says, "At present, white asbestos only is used in

this industry and is imported from Russia and Canada."
Do you see that reference?
A Yeah.
Q Now, this is from the other plant, the other Sigwart plant. There's a reference here to El Maasara, correct?
A Yeah, they speak about a site south of Cairo, where in Madkour they talk about the site being north of Cairo.
Q Right. So in this one, they are referring to the site south of Cairo, which was referred to in Madkour as having an increased incidence of meso, correct?
A Well, in Madkour, they describe Shubra El-Kheima as an industrial city at the northern boundary of Cairo. I'm not sure exactly what you are referring to in terms of south.
Q Oh, I'm sorry. I was looking back at that introduction.
A Oh, okay.
Q We referred to the fact that it says, "Workers employed since 1948 by the Egyptian asbestos company Sigwart at the mills in greater Cairo, El Maasara and Shubra El-Kheima."
A Oh, okay.
Q That's the reference.
A All right.
Q And here they are referring to the El Maasara facility, which there's a reference to being -- having all three

commercial asbestos types, correct?
A Yes.
MR. PFAHL: Let's go off the record for a moment.
(Discussion off the record.)
(Recess from 12:09 to 1:03.)
(Exhibit No. 18 marked for identification.)

MR. PFAHL: Let's go ahead and go back on the record.
Q (By Mr. Pfahl) Dr. Brodkin, I wanted to follow up on a couple of things before I launch into something new.
First of all, your fee for your case review and preparation of a report and testimony, what is that currently?
A My rates are all by the hour. For all preparatory activity it's $550 per hour; a deposition is $650 per hour.
Q And do you have an estimate of the number of hours you've spent up to today in reviewing the material, talking with counsel, preparing your notes and to the extent necessary re-reviewing things to prepare for today's deposition?
A Yes. My medical evaluation was about 43 hours, and I spent about 6 hours preparing yesterday for the

deposition.
Q I had asked you about dose estimations generally -- well, as it related to joint compound. Do you recall that?
A Yes.
Q Is it fair to say that you have not undertaken the task of trying to perform a dose reconstruction estimate for Mr. Quirin for -- on a fiber-per-cc-year basis, so a cumulative basis, to any particular asbestos-containing material or source?
A That's true. Doing fiber cc year reconstructions is not part of my practice, and I have not done it in this case and not for any product entity.
Q And just to clarify, you have not done that for Mr. Quirin as it would relate to any particular branded product, correct?
A Correct.
(Exhibit No. 22 marked for identification.)

Q (By Mr. Pfahl) I'm going to mark as Exhibit 22 a copy of an article that I know you are familiar with. For the record, this is an article by McCoy and others from the Journal of ASTM International, Volume 8, No. 1. It's titled Mesothelioma in Drywall Finishing Workers from 2011.

A Yes, I'm familiar with it.
Q And just by way of background, in the abstract it says, "In this manuscript, we evaluate the context in which workers were exposed to drywall joint compound based on the state of the art of the construction industry during the post-World War II era through the 1970s and conduct a review of the scientific literature associated with the drywall trade and occupational exposure to airborne asbestos from drywall finishing work practices."
A Yes.
Q Do you see where I've read that?
And you have written in conjunction with some other physicians something in the nature of a comment about this article; is that right?
A Yes, I did with some of my co-investigators from CARET did submit a peer-reviewed commentary on this article.
Q I'm going to hand you Exhibit 23.
(Exhibit No. 23 marked for identification.)
Q (By Mr. Pfahl) And I'm sure you will find that to be familiar?
A Yes.
Q For the record, Exhibit 23 is an article by Dr. Brodkin and others titled Discussion on "Mesothelioma in Drywall

Finishing Workers," by McCoy, et al.
It's from the Journal of ASTM International, Volume 8, No. 8. And this was published in 2012, correct?
A I believe this was 2011.
Q And I'm looking at 2012. That's when I printed it off.
A Yeah, it's copyrighted from the Journal of ASTM 2011.
Q So the same year that the McCoy article came out, you and others responded to it?
A Yes, specifically some of my co-investigators at CARET.
Q Right. Dr. Cullen for one?
A Yes.
Q Dr. Balmes?
A Correct.
Q Was Dr. Redlich, was she one of your co-investigators in CARET?
A Yes, Dr. Redlich at Yale is a CARET co-investigator.
Q And Dr. Hammar, who is also here in Seattle?
A Yes.
Q Now, you mentioned that your discussion as it's titled or comment was peer reviewed; is that correct?
A Yes.
Q Was the McCoy article peer reviewed?
A Yes.
Q And that went through the same process as your commentary so far as you know?

A Yes.
Q Let's look at the introduction of your commentary. How do you prefer me to refer to this?
A Well, you can call it discussion, discussion or commentary.
Q All right. In the introduction about, let's see, it's halfway through the sentence after the first citation? It begins, "Our experience."
A Okay.
Q Do you see where I am? It says, "Our experience does not support the authors' conclusion of 'lack of a relationship' between mesothelioma and drywall finishing work. Specifically, among 4,060 asbestos-exposed participants in the Beta Carotene and Retinol Efficacy Trial (CARET cohort), plasterboard workers numbering 136 or 3 percent were defined as a high-risk trade -- along with asbestos insulators, sheet metal workers, plumbers/pipefitters, boilermakers and various shipyard trades -- due to their historic asbestos exposure."
Correct?
A Yes.
Q Then you refer to your CARET study that was published in the American Journal of Industrial Medicine, right?
A Well, there have been numerous publications of CARET, but I do reference the 1996 study, which involved a review of

the cohort in the New England Journal of Medicine and the Journal of the Cancer Institute. And the Journal of the National Cancer Institute is one cited in Reference 3.
Q Well, the one where you are referring to the plasterboard workers as being defined as a high-risk trade is to the 1997 CARET publication, right?
A That's correct, the American Journal of Industrial Medicine.
Q And let me hand over Exhibit 24.
(Exhibit No. 24 marked for identification.)
Q (By Mr. Pfahl) Exhibit 24 is from the American Journal of Industrial Medicine from 1997. The title is The CARET Asbestos-Exposed Cohort: Baseline Characteristics and Comparison to Other Asbestos-Exposed Cohorts, and you are one of the several coauthors of this study?
A Yes. A number of the CARET co-investigators are authors.
Q All right. In the abstract there, it says, "The Carotene and Retinol Efficacy Trial (CARET) was a double-blind, placebo-controlled trial of the daily administration of 25,000 international units of vitamin A and 30 milligrams of B-carotene for the prevention of lung cancer. Of close to 18,500 participants, more than 4,000 were asbestos-exposed men recruited from shipyard and

construction trades at five study centers in the United States."

Do you see that?

A Yes.

Q All right. And the reference in your discussion piece about the McCoy article referred to 4,060 asbestos workers?

A Correct.

Q And that's where we get the reference here as to more than 4,000 asbestos workers?

A Correct, the asbestos exposed cohort, yes.

Q Let me have you turn over to 574 of the CARET article.

A (Complies.)

Q On the left-hand column in that first carry-over paragraph at the top?

A Okay.

Q The first sentence, full sentence, there says, "The trial had followed a total of 18,314 participants for a mean of 4.0 years, when active intervention was halted in January, 1996, because of a higher rate of lung cancer in the participants receiving the study vitamins than those receiving placebo," citing Omenn. That's the 1996 study?

A Correct.

Q Or a publication I should say.

A Correct.

Q And here you stop -- you actually stopped the study because you were getting higher incident rates with the study group than the controls; is that right?

A The intervention was discontinued in 1996. So the follow-up part of the study continued.

Q Sure. You still wanted to follow the participants.

A Sure.

Q But you stopped giving them the vitamins?

A That's right.

Q And did you ever determine an association between the vitamin administration and the higher incidence of lung cancer?

A Well, the intervention participants did have a higher rate of cancers, as well as some non cancer end points, cardiovascular disease. It wasn't statistically different than the placebo group, but given the fact that there was a higher incidence, it would be very unlikely that if we were to have carried out the study longer, that there would have been a benefit. That's why it was discontinued.

I don't think it allows any, although there's been much discussion, as to why anti-oxidant vitamins are not effective. It's not clear that there was a significant difference in adverse effects based on the vitamins. It wasn't statistically different.

Q All right. So it was an observation, and since you didn't find there to be any benefit, you, you meaning you the study participants, decided -- or physicians decided just to halt administration of the vitamins, right?

A Well, we had a meeting of the -- of all the scientific committees of CARET in 1996. We met together and made a decision jointly.

Q Let me have you look with me at Materials and Methods.

A (Complies.)

Q It's on 574, left-hand column, under Materials and Methods and there's Overview of the Study?

A Okay.

Q And I will start with about the third sentence which begins, "The asbestos-exposed cohort was recruited." Do you see where I am there?

A Yes.

Q It says, "The asbestos-exposed cohort was recruited by five CARET study centers located in Baltimore, New Haven, Portland, San Francisco and Seattle." I should say Portland, Oregon, I guess, to distinguish that from Maine. "Potential subjects were identified from multiple sources including clinic medical records, union membership rosters, workers' compensation lists, the US Navy and attorneys." And I will stop there.

To what degree were attorneys involved in

identifying potential study subjects? Do you recall?

A I don't. At the time that recruitment started, which was in the late 80s, I had just started my fellowship in occupational medicine. So I actually joined the study a couple of years later.

I can't give you the breakdown for that. I just don't know it.

Q Well, by 1996 or 1997, had you started to work at all as a consultant in asbestos-related injury, personal injury, cases?

A Well, from 1993, I had been subpoenaed as an expert in various cases. So yes, by 1996 I had.

Q By 1996, had you served as a medical causation expert in injury claims brought by asbestos-exposed individuals against asbestos-containing product manufacturers? Do you recall?

A Perhaps on a few occasions. Most of the cases I would have done by then were workers' compensation cases, but there may have been some. I would really have to look to see.

Q Now, if we look at that same paragraph?

A Yeah.

Q About halfway through the next sentence, which is broken up a bit by semicolons, there's a reference to "occupational asbestos exposure beginning at least 15

years previously." Do you see that reference?
A Yes.
Q All right. Right after that it says, "Asbestos exposure was accepted only if the participant had (1) worked in a trade known to be at high risk of asbestos exposure for a minimum of 5 years at least 10 years previously, or (2) a chest radiograph that demonstrated changes consistent with asbestos-related disease and an occupational history consistent with substantial asbestos exposure. CARET defined high-risk trades as asbestos insulators, sheet metal workers, plumbers/pipefitters, plasterboard workers, boilermakers, shipyard electricians, ship scalers and ship fitters," end quote.
All right. And so those were the high-risk trades?
A Those were the designated high-risk trades, yes.
Q And were you involved in defining the designated high-risk trades at all?
A No. The parameters for entry into CARET, again, would have been in the late 80s before I started my fellowship. So no.
Q Do you know what the -- what criteria were used to designate a trade as a high-risk trade?
A My understanding is that it was based on historical evidence of significant asbestos exposure in that trade. The purpose of CARET was to recruit individuals at high

risk for developing cancer either by virtue of smoking or having heavy asbestos exposure. So there would have been either medical industrial hygiene evidence or both that these were individuals with a significant history of asbestos exposure during those historic periods.
Q Did the criteria to be determined or to be defined as a high-risk trade require that there be occupational epidemiology demonstrating an increase in lung cancer or other types of cancers for those groups, those cohorts?
A I don't think it was based on a particular algorithm that required an epidemiologic study. It was based on the experience within those trades.
Now, it could include epidemiology, but it would include industrial hygiene and medical -- occupational medicine experience as well.
Q Looking at the tables on 577, Table 4 indicates High-Risk Trade Distribution: CARET Asbestos-Exposed Cohort, and that's the distribution of the various trades amongst the five study centers?
A Correct.
Q And then Table 5 is Distribution of Parenchymal and Pleural Abnormalities: CARET Asbestos-Exposed Subjects, correct?
A Correct.
Q And here the chest radiograph results are not broken out

by trade, correct?
A That's correct.
Q So it would just be the study center that would have a certain percentage that was identified, for example, positive parenchymal results, right?
A That's correct, based on the x-ray readers.
Q And the 1997 study refers to results from the chest radiographs. It also refers to pulmonary function results; is that right?
A Yes.
Q Respiratory symptoms, correct?
A Yes.
Q And, again, that would be based upon study center location, not the individual trades; is that right?
A To my knowledge, respiratory symptoms, I have investigated and published on respiratory symptoms in the CARET cohort, there's never been a breakdown by trade to my knowledge.
Q In this particular article about the CARET cohort, there's no reference to any of the study subjects involved in the high-risk trades as having experienced mesothelioma; is that right?
A Well, you couldn't enter CARET if you had a history of preexisting cancer. Basically, this was a study to prevent initial cancer, so that would have been an

exclusion criteria.
Q Right. So if you had lung cancer -- already had lung cancer or mesothelioma, you wouldn't be in this study?
A Right.
Q On the last page, 580, on the left-hand column, the second full paragraph, "There are several limitations," do you see where I am?
A Yes.
Q "There are several limitations to the use of this cohort to study the natural history of asbestos-related lung disease. First, there exists the potential for respondent bias in terms of misclassifying the extent of asbestos exposure in this cohort of men who volunteered for a prospective randomized chemoprevention trial to reduce the incidence of lung cancer."
And what is it about the nature of the study that might result in respondent bias about misclassifying asbestos exposure?
A Well, if there had been inaccurate reporting of their occupational history and, you know, a desire to enter the trial, an active intervention trial to prevent cancer, could it have influenced responses. I think it's a discussion of that.
Q Oh, okay. So people may want to get into the study when they hear something might involve cancer prevention?

A Yes. I mean, there could be a potential bias.
Q With respect to your discussion of the McCoy paper then, the fact that you are referring to these high-risk trades as including plasterboard workers, that was as was defined in your article from 1997, right?
A Correct.
Q And it was a function of asbestos exposure or potential exposure as opposed to somebody who's in a known cohort with an increased incidence rate for lung cancer, right?
A Well, again, these were individuals that by virtue of their work were recognized as having high historic exposures to asbestos. That's how they became participants in CARET.
Q You indicate in the discussion, it says, "At the time of the initial analysis in 1996, 23 mesothelioma cases had occurred in the CARET cohort." Is that correct?
A Yes, that's correct.
Q So these are cases where mesotheliomas are developed among the study participants following their initial entry into the study?
A Correct. They would be incident cases where they developed during the course of the study.
Q And out of the 23 cases, you said one case was among a plasterboard worker; is that right?
A Well, as of 2011 when this article was written, there's

been a longer follow-up of the CARET cohort. There are now 40 cases of meso within the CARET cohort. It was one of those 40. I can't tell you if it was one of the 23, but it's one of the existing 40 cases.
Q Oh, I'm sorry. And you did mention that, and I misread it. It says with 40 total cases, currently one includes -- is a plasterboard worker; is that it?
A Correct.
Q And I believe you told me in a previous deposition that you weren't in a position to state whether or not plasterboard work was that person's only exposure to asbestos, correct?
A Correct.
Q In the next paragraph, you refer to the Stern study; is that right?
A Yes.
Q And we've talked about that before. That's the plasterer cement mason cohort?
A Correct.
Q All right. Do you agree with the representation by McCoy on Page 5 of the McCoy article at Table 1 where there's a reference to that Stern study, and there is a "measure of effect" column which indicates there are four observed mesothelioma deaths with a PMR equal to 188? Do you see where I am?

A Yeah, you read it correctly.
Q Okay. And there's a footnote there that says, "Expected rates for mesothelioma were unavailable. PMR was not statistically significant. Two of the four mesothelioma deaths were in plasterers."
Do you see the footnote, first off?
A I do, yes.
Q Do you agree with McCoy's representation that the PMR was not statistically significant?
A Well, yes, but it's not -- I don't think it's a complete representation of the Stern study. The Stern study analyzed -- it's correct that it didn't do a PMR analysis of mesothelioma only. It did a PMR of all respiratory cancers that was statistically significant. So that's what the Stern study found.
The other thing is that while there were four cases of mesothelioma derived from I believe it was the pathologic data, the nosologists review of the death certificates indicated likely 40 mesotheliomas, and that discussion is not represented in McCoy's article.
Q And with respect to nosologists would be the death certificate statements or findings, I guess?
A Yes, it would be the death certificate findings.
Q All right. And the calculation that Stern did, a PMR, though, was based upon the pathological findings; is that

right?
A I believe so, yes, and the PMR was 188.
Q In your discussion of McCoy, you also refer to Fischbein's study, correct?
A Yes.
Q And that was the second of two studies -- well, the second of two articles where the Mount Sinai group followed some professional drywallers, right?
A Yes.
Q So you had Rohl as the lead author in the first article that was published, which was a fiber release and exposure study?
A Yes, a Science 1975 study.
Q And then in 1979, Fischbein's article came out, and that discussed some parenchymal abnormalities; is that right?
A That's right.
Q Among other things? They also had the exposure data?
A They also re-summarized the exposure data from the earlier study.
Q And do you recall if Fischbein controlled for other potential causes of parenchymal abnormalities that may be read on a chest x-ray?
A Well, the design was a cross-sectional study, and they had readers who did an ILO classification. I don't think there was an analysis beyond that.

Q In your discussion of McCoy and today you've also mentioned the reference to the IARC publication?
A Yes.
Q And in particular the 2009 publication and its statement that all of the various asbestos types cause mesothelioma, right?
A Yes, all the major commercial fiber types.
Q All right. As you sit here today, do you recall what epidemiological studies that IARC relied upon or indicated that it relied upon to make a finding of chrysotile's relationship with mesothelioma?
A The IARC position paper published their conclusions based on their review of the evidence. They didn't cite specific epidemiologic studies. Their conclusions were based on the totality of the scientific evidence, but it wasn't a review or citation of individual studies.
Q Do you recall if IARC indicated what its standard was or whatever standard it applied in making a statement about chrysotile and its ability to cause mesothelioma?
A They -- they cited it as a known Group 1, a human carcinogen for all fiber types. Their process of doing that is published through the World Health Organization in terms of what a Group 1, Group 2, Group 3 carcinogen is, possible, probable or known human carcinogen. It's based on a weighting of the evidence. But you would have

to refer to the specific IARC publications for that.
Q And you don't recall what -- if they have different weights assigned to human epidemiology versus in vitro studies versus animal studies, for example?
A IARC would consider all of that. They would consider the toxicological data, as well as the epidemiologic data, as well as the mechanistic data.
Q Do you know if they would -- do they rate it differently though?
A Well, based on -- based on the data, for example, if there's toxicologic evidence that a substance is carcinogenic but not human evidence, it's not going to be a known human carcinogen. It may end up on the possible list based on toxicologic data alone. But there would have to be more. There would have to be human data to be a known human carcinogen.

(Exhibit No. 25 marked for identification.)

Q (By Mr. Pfahl) I'm going to hand you another article. I will pass that over. This is titled Re-Creation of Historical Chrysotile-Containing Joint Compounds by Brorby, B-R-O-R-B-Y, and others. It's from Inhalation Toxicology, Volume 20, 2008.
Dr. Brodkin, let me have you just look at that and

see if you are familiar with this article at all.
A (Peruses documents.) Well, I can tell you just sitting here, I don't have a working memory of it. It's not to say I haven't seen it before. But, I mean, if you have specific questions, you can certainly guide me to them.
Q Sure. First of all, the authors Brorby and Sheehan are from Exponent, as is Greene. Are you familiar with Exponent?
A Yes. I mean, I can't say I have worked with them, but I have heard of them, sure.
Q And what is your understanding of Exponent?
A Well, I mean, I can't give you a comprehensive description of Exponent. But they certainly provide professionals experienced in toxicology, industrial hygiene, occupational medicine-related disciplines to address health-related questions. But I can't give you any more specifics about that.
Q Fair enough.
Do you know if they are involved in providing expert consulting in litigation like you do?
A I have seen their name associated with that. I have seen Exponent reports in association with litigation in the past.
Q And Aeolus, the reference to Berman, are you familiar with that company at all?

A I would say not.
Q And then the last author of this is Holm, and he works for Georgia-Pacific? Do you see that?
A I see it, yes.
Q And in the -- under the Introduction, there's a reference underneath there toward the bottom that says, "This research was primarily funded by Georgia-Pacific LLC who has been in litigation related to joint compound"?
A Oh, where is that?
Q Oh, I'm sorry. I didn't do a good job of pointing that out. (Indicating.)
A Yes, I see that.
Q All right. And in 2008, you were certainly aware that Georgia-Pacific had been involved in asbestos-related personal injury litigation, correct?
A Yes.
Q And you have been involved in some of those cases, correct?
A Yes.
Q And so in looking at this paper, based upon Mr. Holm's involvement and the funding from Georgia-Pacific, it wouldn't be a surprise to you that this would be a study that was being prepared in part to help defend against litigation, right?
A That would likely be my assessment based on what has been

stated here.
Q Now, would that affect your review of the study?
A It wouldn't affect the review of the study. I mean, I would review the study for its own merits and methodology. You know, I mean, if there were methodologic problems --
Q Sure.
A -- it can raise a question in terms of the funding source. But, no, the study has to stand alone, and that's independent of the funding source.
Q All right. And so -- right. The science has to be sound regardless of how it's funded?
A Right.
Q Whether it's NIOSH or Georgia-Pacific, right?
A True.
Q And Inhalation Toxicology is a peer-reviewed journal, right?
A Yes.
Q And so peer reviewers are going to read this and take a look at the underlying scientific methods and protocols, correct?
A That should be the process.
Q And you've done peer review yourself, correct?
A Often, yes.
Q And so that's something that you do as you are interested

regardless of who the authors are is reviewing it and finding out if the methods that were employed are ones that are known to be reliable or that the protocol that is used appears to be reliable; is that right?
A Correct. You would assess for reliable and valid methodology.
Q Let me have you look at the abstract with me. This says, "Chrysotile-Containing joint compound was commonly used in construction of residential and commercial buildings through the mid 1970s. However, these products have not been manufactured in the United States for more than 30 years." Right?
And you agree with that? By 2008, it had been about 30 years or more since asbestos-containing joint compounds were being manufactured and sold; is that right?
A Yeah, I think that would comport with my understanding.
Q It says, "Little is known about actual human exposures to chrysotile fibers that may have resulted from use of chrysotile-containing joint compounds because few exposure and no health effect studies have been conducted specifically with these products."
Do you agree with the author's statement there?
A No, just on the basis of that statement alone. Obviously I haven't read the whole article, or if I have, you know,

I don't recall it. So I'm just speaking to the line itself.
Certainly by the time this was published, everything -- you know, the publications I have cited from the 1970s would have been known to the authors that certainly document exposure associated with chrysotile-containing joint compounds. So I don't agree with that statement.
And then in terms of health effects, just, again, reacting to the line --
Q Sure.
A -- I don't know the context of the article, certainly the Fischbein study would have been known, and certainly Stern, Lehman, Reuter, Leigh, Rodelsperger, those studies would have been known, certainly in terms of lung cancer risk, Journal of Occupational Medicine in 1976 would have been known. So I don't really understand the statement "no health effect studies have been conducted."
Now, maybe they are referring to, you know, a specific product as opposed to joint compounds in general, but I wouldn't agree with the statement as it would relate to joint compounds in general.
Q Let's look at the next sentence. It says, "Because limited amounts of historical joint compounds were available and the stability or representativeness of aged

products suspect, it is currently impossible to conduct meaningful studies to better understand the nature and magnitude of potential exposures to chrysotile that may have been associated with historical use of these products. Therefore, to support specific exposure and toxicology research activities, two types of chrysotile-containing joint compounds were produced according to original formulations from the late 1960s." And I will just stop there.
Do you have any disagreement with the author's statement that there's too little historical samples of joint compound out there to really be of value for studying its exposure and effects?
A Boy, that I can't really speak to. Yeah, it's not an area that I have looked at. Again, I don't -- in terms of their sentence, I don't understand why they are saying there should be an issue about the nature and magnitude of potential exposure to chrysotile from joint compounds because I think that was studied in some depth from the 1970s and forward.
So I'm not sure why they have the need to do this, but in terms of there actually being the joint compound, I can't speak to that.
Q All right. So you think that the studies that have been published up to 2008 firmly establish that exposure to

chrysotile solely from joint compound products causes mesothelioma in humans?

A Well, I think the studies indicate there's a significant risk associated with working with joint compounds. And, of course, I would cite the studies on my reliance list.

Now, does that mean there couldn't have been additional exposures within an occupational setting? That's possible. I mean, a number of these studies have spoken about that.

But, for example, certainly Leigh in American Journal of Industrial Medicine identified plasterers as a high-risk trade using joint compound, but they may have used other materials as well.

So, you know, this is an issue of occupational activity. Someone who was restricted just to drywall would be a much smaller group like Fischbein's study of drywallers.

Q And there are far fewer studies of cohorts that are just drywall workers, right?

A Correct.

Q And where you have them, as you have said before, like in the Robinson study and the Wang/Dement study, they are fairly limited in number, aren't they?

A That's right. They are going to be much smaller groups which for a disease like asbestosis certainly may be

amenable to looking at disease states like asbestosis, but for a rare disease like mesothelioma, it's not going to offer a very powerful study.

Q And in fact in Robinson and Wang, they had groups of -- or cohorts of drywallers, but they didn't have any mesotheliomas in those groups, though, that doesn't surprise you, right?

A Right. The studies really weren't well designed to look for a rare disease outcome like that.

Q You need a lot more people, don't you?

A Correct.

Q Let me read on after that, the end of that sentence that says from the late 1960s. Do you see where we were?

A Yeah.

Q It says, "To the extent possible, ingredients were the same as those used originally with many obtained from the original suppliers. The chrysotile used historically in these products was primarily Grade 7RF9 from the Philip Carey mine. Because this mine is closed, a suitable alternative was identified by comparing the sizes and mineral composition of asbestos structures in a sample of what has been represented to be historical joint compound (all of which were chrysotile) to those in samples of three currently commercially available Grade 7 chrysotile products. The re-created materials generally conformed

to original product specifications (e.g. viscosity, workability, crack resistance), indicating that these materials are sufficiently representative of the original products to support research activities."

And do you see where I have read that?

A Yes.

Q All right. Now, let me ask you about their substitution of a Grade 7 chrysotile that's not from the Philip Carey mine. I believe, as we talked about before, looking at exposure and then even a physiological response to dust from an asbestos-containing product, it wouldn't matter to you if they used Philip Carey, Johns-Manville, Calidria, Brazilian chrysotile or chrysotile from Balangero, right?

A In terms of health effects, it doesn't bother me that they would use other chrysotile. I mean, the Grade 7 indicates that these may have had a propensity for shorter fibers, although, there's still a very wide range of lengths.

So trying to standardize for length of fiber may be somewhat of an issue. But the fact that it's chrysotile doesn't bother me, per se, from one mine versus another.

Q Let me have you turn over to 1044. Just a couple of questions there.

A (Complies.)

Q What I was going to ask I think we've covered because they substituted in a chrysotile, but it was of the same grade that was used in historical chrysotile samples, right?

And then you understand from the -- at least the abstract that we've read is that the intention here was to reconstitute a product for further study?

A That seems to be the goal, yes.

Q Okay. And you haven't -- or you just don't recall reading this in its entirety, but do you have -- based on your recollection, do you have any disagreement with the author's representation that the recreated materials generally conformed to original specifications such that they thought it was sufficiently representative of the original to support their research activity?

A Well, I mean, I understand that that's their goal. I would say this is probably a little bit outside my area as a physician because it really has to deal more with material science, and that's not my area.

I mean, I'm reading with interest what they say about the health effects, and I don't agree with them. But in terms of the technical issues of recreating this, it's really not my area of expertise in terms of the physical properties, but they certainly are discussing their approach.

Q To your knowledge have other companies funded research that have looked at health effects from a particular chrysotile-containing product?
A The studies that I have seen in the epidemiologic literature have not spoken to product-specific information. I know that the Rohl study looked at, I believe, 25 different products, but they don't discuss, you know, product specific exposure measurements.
Q Are you familiar with asbestos-containing material or product manufacturers that have funded studies whether it be just chrysotile studies or other types of studies that relate to asbestos-related disease?
A The one I'm most familiar with is, well, Soule in the Gypsum Association, 1973. I mean, that was an industrial hygiene study to look at exposure measurements, but it certainly was addressing health concerns.
(Exhibit No. 26 marked for identification.)

Q (By Mr. Pfahl) Dr. Brodkin, I'm going to hand you Exhibit 26. This is an article from Bernstein and others. It's Inhalation Toxicology or published in Inhalation Toxicology from 2008 as well. The title is A Biopersistence Study Following Exposure to Chrysotile Asbestos Alone Or in Combination With Fine Particles.

And my first question is does this look like an article that you have read before or studied before?
A I may well have seen it. I don't have a working memory of it as I sit here. So, again, I would have to -- I'm glad to comment on specific lines, but it's not from a working memory of it.
Q All right. Sure.
And if we look at the reference to Inhalation Toxicology, Volume 20 -- if you still have that handy?
A Yes.
Q You will see that that's the same publication as the Brorby article we just discussed?
A The same volume, yes.
Q The same volume. Right.
And this study would have gone through peer review as well, correct?
A Yes, Inhalation Toxicology is a peer-reviewed journal.
Q Do you know or -- well, first of all, do you know David Bernstein?
A I know the name, but, no, not personally.
Q Are you familiar with the nature of his work?
A You know, I really couldn't speak to that. I haven't researched it, I mean, other than what they've indicated, that he's a consultant in toxicology.
Q You see that Holm is another one of the authors here?

A Right.
Q Just like he was with the other paper?
A Right.
Q And Georgia-Pacific is identified as his employer?
A Yes.
Q And down at the very bottom of the left-hand column, there's a reference that says, "This research was sponsored by a grant from Georgia-Pacific"? Do you see that?
A I see it, yes.
Q And so, again, you understand that Georgia-Pacific is funding this study, right?
A That appears to be the case.
Q All right. And it would be your assumption based upon the involvement in the company and even one of the authors who was employed by them that this is likely done in some respects in order to defend against asbestos personal injury litigation?
A Well, they don't state that here. So, I mean, I guess I can't really speak to that. I mean, it's possible it could be used for that. I just don't know enough about this article to really speak to that.
Q Sure. Let's look at some of it and see if it helps, and if there are areas which you feel that you are not ready to comment on, just tell me. All right?

A Okay.
Q As background, let's look at the abstract. It says, "In designing a study to evaluate the inhalation biopersistence of chrysotile asbestos that was used as a component of a joint compound, a feasibility study was initiated to evaluate the short-term biopersistence of the chrysotile alone and of the chrysotile in combination with the sanded reformulated joint compound.
"Two groups of Wistar rats were exposed to either 7RF3 chrysotile," which is identified as Group 2, "or to 7RF3 chrysotile combined with aerosolized sanded joint compound (Group 3). In addition, a control group was exposed to filtered air."
Do you see how I muddled through on that particular --
A You have read it correctly.
Q All right. You are being kind.
So do you understand generally that this is an animal exposure -- inhalation study?
A It appears to be, yes.
Q And have you done animal inhalation studies yourself?
A No. I've relied on them in terms of understanding the biologic and toxicologic evidence for disease, but I don't conduct animal studies personally.
Q All right. But you've read and you are familiar with

animal studies?
A Yes, and I do rely on them.
Q All right. And they do inform your opinion about biological plausibility or biologically plausible sequelae to exposures to things like asbestos?
A Yes, and other substances, yes.
Q Sure. Let's take a look at the introduction. It says, "The inhalation biopersistence study was originally developed in order to quantify the rate at which fibers clear from the lung." And then there's a reference to Bernstein and Musselman.
"Since its inception, numerous synthetic and natural mineral fibers have been evaluated using this protocol design," referring to Bernstein and Reigo. "For synthetic mineral fibers, the biopersistence of the fibers longer than 20 microns was found to be highly correlated with pathological response in chronic inhalation studies and with tumorigenic potential in chronic intraperitoneal injection studies. More recently, commercial chrysotile evaluated under the same protocol was found to clear rapidly from the lungs with clearance half-times of fibers longer than 20 microns ranging from 0.3 to 11.4 days depending on chrysotile fiber type. However, these studies always involve the exposure of the pure bulk product without the addition of

any secondary aerosol to the test atmosphere." And I will stop there.
The animal studies that you are familiar with that look at a biological response to asbestos, those have been injection and intraperitoneal area or they would include --
A Well, they include injection studies either into the pleura or peritoneal membranes or they can be through an inhalation methodology.
Q Right.
Would you agree with the authors that those types of studies used pure bulk chrysotile as opposed to chrysotile and some other particulate?
A Most of the toxicologic studies that I'm aware of would study a pure fiber type to compare with other fiber types or controls. So it wouldn't typically be a mixture. Now, I can't say that in all cases, but most studies are designed that way.
Q All right. The next paragraph states, "We set out to evaluate the biopersistence of the commercial chrysotile asbestos that was used through the mid-1970s in a joint compound intended for sealing the interface between adjacent wall boards. An inhalation biopersistence study was designed to include not only the chrysotile component of the joint compound alone but also the sanded

particulate component, as real-life exposure to abraded wall board would result in concomitant exposure to both chrysotile fibers and joint compound particles. The inclusion of the sanded particulate fraction was considered important in order to determine whether any of the particular components of the joint compound influenced the biopersistence or pathological response to the chrysotile."
Do you see where I have read that?
A Yes.
Q And you understand that what they want -- the hypothesis here is to see whether or not the asbestos dust -- well, the dust that's created from sanding asbestos-containing joint compound and that would be inhalable, whether or not that would be biologically different than just inhaling chrysotile fibers?
A I understand that that is a hypothesis that is being looked at, yes.
Q All right. So do you agree with them that if you are interested in determining whether or not inhaling joint compound dust that would consist of particles of a joint compound in addition to just chrysotile, that combining the two and comparing it with just pure chrysotile would be an important step to determine whether or not you have a biological distinction?

A Well, I understand the nature of the question. You know, in terms of the study design, again, this isn't my area in terms of animal toxicology. I mean, there are a lot of issues that go into using a mixture instead of a pure fiber to make sure that it's not confounded by the total particulates in some way.
But I understand the question. I mean, you know, in terms of the study design, one wants to look at a mixture of materials compared to the pure fiber type and see if there's a difference. So I understand that.
Q All right. And so you can understand what they want to try to accomplish here in terms of a hypothesis and seeing what the results say?
A Yes. I mean, I think they have expressed that.
Q Let's go ahead and look at the methods.
A (Complies.)
Q You see where the methods are in the left-hand column of 1010?
A Yes.
Q And then there's a reference Sanded M971/974 powder? Do you see that reference there?
A Yes.
Q And before it talks about the chrysotile. We discussed that when we were looking at the reformulation study. If you just want to take a moment to refresh your

recollection. But they didn't have Philip Carey chrysotile?
A Right.
Q So they ended up using Johns-Manville 7RF3 chrysotile?
A Right.
Q They consider that to be the most similar to the Carey chrysotile, right?
A I think that was the objective.
Q And then, again, let's look to the next column. The first full paragraph there says, "The reformulated compound was applied according to the instructions of the original material to pieces of drywall, the ends of which were sealed with tape. A notched trowel was used for application. The material was allowed to dry for at least 48 hours and then sanded. Individual boards were sanded for approximately 20 to 30 minutes. Four different boards were needed to obtain sufficient mass of material for the studies. Aluminum oxide medium 120-grit sandpaper was used to generate the sample. The sanded material was collected in a large Ziploc bag, and the bag was sent off for study." This study, right?
A Uh-huh, yes.
Q All right. So you can see what they are doing is they reformulated the joint compound, they put it on a wall, they sanded it and gathered up the dust?

A Yes, I think that's fair.
Q All right. If you want to turn over to Page 1012 with me?
A (Complies.)
Q Animal Exposure, left-hand column. This will just give us some -- a reference here. It says, "Three groups of laboratory rats (groups 1, 2 and 3) were be exposed," it should have "sic" put in there, "for six hours per day for five days."
Do you see that reference?
A Yes.
Q So I will read it without their typo. "Three groups of laboratory rats were exposed for six hours a day for five days"?
A Right.
Q Group 1 filtered air, group 2 a fixed-exposure to well-characterized fibers of chrysotile 7RF3 and group 3 a fixed-exposure level of well-characterized fibers of chrysotile 7RF3 mixed with sanded powder.
A Yes.
Q Correct?
And that will just give us a reference. I want to move along.
Let's go to Page 1015, left-hand column under results. "Validation of lung digestion and counting

procedures," do you see where I am?
A Yes.
Q I see you flipping over. Are there things you want to review?
A Well, I haven't -- I don't have a working memory of this article, so I'm trying to get through it. I mean, I'm certainly looking at what you are reading.
Q Okay. Under the Results section Validation of the Lung Digestion and Counting Procedures, it says, "Validation of lung digestion and counting procedures is essential to the legitimacy of this type of study, although it was often absent from early studies. Such validation provides confidence that there is no significant alteration of the fiber length or distribution during fiber recovery." Let me just stop there.
Are you familiar enough with this type of study to understand what they are referring to from a validation standpoint?
A Well, again, I don't as part of my practice conduct lung digestion or counting. So from this technical aspect, it's not really my area.
Q Let me refer to the next paragraph. It says, "The only method suitable for validation of chrysotile fiber recovery would be a parallel analysis using a noninvasive measurement technique such as confocal microscopy and

comparison of these results with those obtained following transmission electron microscopy examination of the digested lungs. This type of comparative analysis of the fiber number and size distribution is planned for the main study. Previous studies using the same procedures used here have confirmed that the fiber recovery procedures do not significantly alter the fiber size distribution or number of the chrysotile fibers."
And then there are references to three other Bernstein studies. Do you understand what they are referring to with respect to the confocal microscopy?
A No. This really is not my area.
Q All right. I just wanted to see.
A I mean, I understand, you know, the concept of microscopic analysis and, you know, transmission electron microscopy and the limitation of those modalities, but it's not something I perform as part of my practice.
Q No, I understand. And you don't know -- well, do you know whether or not confocal microscopy can take 3D -- essentially, like, 3D images and be able to pick out fibers in a particular space?
A No, I can't speak to that.
Q Okay. Fair enough.
Turn over to 1017 with me.
A (Complies.)

Q Down in the bottom of the right-hand column, there's a heading Lung Fiber Burden. Do you see that?
A Yes.
Q And the paragraph which begins on 1017 and then carries over states, "The number, concentration and size distribution of the chrysotile fibers and lungs of the rats from group 2 chrysotile exposure," and I'm over on to 1019 now, "and group 3 chrysotile and sanded component immediately after the termination of a five-day exposure defined as day zero, and at three days following cessation of exposure, day three, are presented in Tables 5 and 6 respectively. The data for each animal individually and group meanings are shown for each parameter. As shown in Table 5 immediately following cessation of exposure zero days, there was a mean of 27.2 million WHO fibers remaining in the lungs of the rats in group 2, which received only chrysotile. Rats from group 3, which received both chrysotile and the sanded powder, had only 2.3 million WHO fibers at the same time point, zero days. Similarly, there were a mean of 0.44 million fibers longer than 20 microns in length from rats of group 2 and a mean of 0.037 million fibers longer than 20 microns in length from rats of group 3. Across all size ranges, there was approximately an order of magnitude difference in the mean number of each size category of

fibers remaining in the lungs of group 2 as compared to group 3."
Do you see where I have read that?
A Yes.
Q All right. And so they are reporting that at the measurement points that they have, zero days and three days post exposure, the fibers remaining in group 3, the combined exposure rats, was an order of magnitude approximately less than the fibers that remained in the chrysotile only rats; is that how you read that?
A They have reported that, yes, as their observation.
Q Let me have you turn over to the discussion on 1025. The right-hand column.
A (Complies.)
Q You see it's Biopersistence of the 7RF3 Chrysotile is the heading?
A Yes.
Q The second paragraph there says, "In the current comparative study, identical fiber aerosol generation systems were used for each exposure group with the only difference being that in group 3, a separate aerosol generator produced an aerosol of micronized sanded material that was added in the airstream to the chrysotile aerosol. The aerosol fiber number and size distributions were similar in group 2 and 3 as presented

earlier.
"Care was taken in the experimental design to ensure that the fiber number and size distribution of the chrysotile of the aerosols of the two exposure groups were similar. Therefore, based upon the exposure aerosols and the dynamics of lung deposition, the number and size of fibers deposited in the lungs was expected to be similar in both groups. However, the numbers of fibers remaining in the lungs at both the first and second sacrifice time points, immediately after the end of exposure and three days later, were quite different between the two exposure groups."
All right. Do you see that?
A Uh-huh.
Q They are referring to that order of magnitude difference that we saw, right?
A Yes.
Q It says, "Influence of the sanded aerosol on fiber clearance. As has been described previously, chrysotile fiber clearance is related to the interaction of the mineral structure of the fiber with lung environment and cells." Bernstein and Hoskins from 2006. "Long fibers with low biosolubility are the most pathologically active fiber size category, and clearance of these fibers is very slow due to the inability of macrophages to

phagocytose them completely, leading to a frustrated phagocytosis." Let me just stop there.
Do you agree with the reference that low biosolubility long fibers are very slow to clear the lungs?
A Fiber length can be a factor in clearance.
Q And can biosolubility be a factor?
A Yes.
Q So if you have long amphibole fibers, you expect their clearance to be much longer than that for short chrysotile fibers, correct?
A That's well known, and certainly half-life in the lung is different. In other systems, the pleura, it's different, but in the lung, that's true.
Q Let me read on here. "Chrysotile is a rolled silicate sheet with magnesium on the outside of the sheet and silica on the inside." I will stop there.
Do you agree with that description of chrysotile?
A Well, I'm aware that chrysotile is a hydrated magnesium silicate. They are getting into some fairly technical physical structural differences that are a little bit beyond my expertise. I would say that's more of a mineralogic physical issue.
Q Okay. Fair enough.
It says, "Longer chrysotile fibers are shortened in

the lungs, and this is considered to be a consequence of the action of the lung environment on the chemical structure of the chrysotile fibers."

Do you see that?

A Yes.

Q Do you agree with that statement generally?

A Yes. I mean, I think it's been well demonstrated that there is a difference in kinetics with chrysotile in the lung.

Q The next sentence says, "The dual action of both the lung surfactant and the acid environment of the macrophage phagolysosome may leach the magnesium layer and break apart the silica bonds. This causes longer fibers to break into smaller fibers and particles which can then be fully phagocytosed and cleared by the macrophage."

Do you see where I read that?

A Yes.

Q Would you agree with that description?

A Well, certainly macrophage clearance is one mechanism of clearing fibers.

Q All right. And do you know if chrysotile fibers break down in the lung surfactant?

A I mean, that's a pretty specific physical question that I can't say that I've studied to any extent.

Q You defer to someone like a Dr. Brody, for example?

A Right. I mean, someone who has basically modelled this type of experiment and measured half-lives within the lung would be better able to answer that question.

Q Let's look over on the right-hand column, Macrophages and Biopersistence. Do you see where I am?

A Yeah.

Q It says, "The finding that the total number of fibers present in the lungs of the animals in group 3 (chrysotile and sanded material) is approximately an order of magnitude less than in group 2 (chrysotile alone) is intriguing. This difference was evident immediately at the end of the five-day exposure period suggesting that there had been accelerated clearance during ongoing exposure to the combined fiber/particle exposure."

Would you agree that the findings that they had do suggest that there was some type of an accelerated clearance for the combined exposure rats compared to the chrysotile-only rats?

A Well, I think the observation they are discussing is that there is evidence of a change in clearance between the two groups. I think, you know, the issue in the discussion is why. And I guess I'm a little uncomfortable speaking to that because, again, we're outside my area. But, I mean, there would be all sorts

of questions as to the rat's response to a different particulate load, what could be the confounding factors, is it a true difference in clearance. I mean, these are areas that are outside my area really.

Q Let me ask you about the next paragraph that begins, "In seeking." Do you see where I am?

A Yes.

Q It says, "In seeking an explanation for this rather paradoxical finding of increased clearance of fibers during concomitant exposure to particles in group 3, we noted that histopathological examination of the lung showed increased macrophage numbers in group 3 as compared to group 2. The pathologists reported that the increased number of alveolar macrophages in group 3 may be considered as a reaction to the inhaled fiber powder mixture."

Do you see where I have read that?

A Yes.

Q And so what they did recognize is there was an increased macrophage response in the mixed particle, correct?

A Right.

Q And can that help explain the more rapid breakdown of chrysotile fibers in your opinion?

A Well, I think they have offered another hypothesis. I mean, I don't think -- I mean, they are positing that

that could be a possible explanation. I'm wondering if they have all the controls they need to really make those kinds of conclusions.

I mean, they have one group with a mixture and one group with pure fiber. I would like to see some other controls with other mixtures to really sort of speak to what the actual mechanism is. But, I mean, they are discussing possibilities here.

Q And your preference would be to know whether or not if you had chrysotile and some other kind of particulate as a combined exposure?

A Exactly. I mean, there seems to be a conclusion here that if you put this particular mixture that would be in joint compound and inhale it into the lungs, that somehow the clearance is affected.

It raises the question, well, is that unique to joint compound? Is there something going on with the mixture or the physical reaction? I mean, choosing your controls is important. I mean, these aren't the kinds of experiments I do, but it's something one wonders about.

Q Let's look at the bottom of the paragraph beginning with the sentence, "The fact that there is greater." Do you see where I am? It's about seven lines up from the bottom.

A Oh, okay. (Peruses documents.) Yeah.

Q It says, "The fact that there is greater clearance of the long chrysotile fibers in the combined exposure group means that there must be more rapid leaching and breakage of these long fibers as the macrophages intend to engulf them. The accelerated disintegration of the chrysotile fibers in the lungs of the rats receiving the combined exposure could be explained by two factors: The increased numbers of macrophages present in these lungs and the macrophages could have a greater ability to cause leaching and breakage. The greater numbers of macrophages mean that the total amount of long fibers inside macrophage phagosomes is increased, and so the total 'acid stress' applied to these chrysotile fibers is greater. The reasoning that there is greater potential for individual macrophages to cause disintegration is supported by the finding of a mild degree of inflammation, as indicated by the increased numbers of macrophages seen in the lungs of the combined group. Exposure to particles leads to the induction of inflammation," reference the Silver, Schaible, Haas, "and a macrophage infiltrate typical of inflammation was documented in the lungs of the combined exposure. During inflammation macrophages undergo 'activation,' a change in differentiation status to more active secretory and functional phenotype. Of special relevance here is the

finding that phagosomes of active macrophages rapidly become more acidic than those of resting macrophages by about 1 pH unit," citing Schaible.

Do you see where I have read that?

A Yes.

Q Okay. And here they are talking about the increased number of macrophages and also an increase in total acid stress in the area, right?

A Yes, that is discussed.

Q And do you believe that that is a biologically plausible explanation for why there can be increased fiber dissolution in the combined group as opposed to just the chrysotile only group?

A Well, I think it's a reasonable discussion of possible mechanisms. I don't think this experiment is designed to really answer the question. I think it's a discussion of possible mechanisms. I think in that context, it's a reasonable discussion. But I don't know that this study is really designed to provide any specific answers.

Q But from a biological standpoint, having more macrophages and macrophages that are more activated could explain the greater -- the greater chrysotile fiber clearance that was seen in the combined group; would you agree?

A It could, yes. That could be a mechanism. But, you know, I don't want to overgeneralize about this study,

and I don't think perhaps the authors have.

But, I mean, I come at these articles from a health perspective, and, you know, it begs the question you want to see, well, you know, what was the impact on the lung, was there evidence of tissue injury, fibrosis, because increased inflammation can result in increased injury as well.

But this study is very limited to clearance. So I think there's a discussion of it. And I don't see a problem with that, but I don't think the study is necessarily designed to really answer why they see this change in clearance.

Q But they did provide some observations?

A Sure.

Q And one of the observations was increased clearance in the combined group, right?

A Yes.

Q And they also had the observation that there was a greater macrophage response in the combined group?

A True.

Q And they had -- that was -- strike that.

MR. PFAHL: All right. Let's go off the record for a minute.

(Discussion off the record.)

(Recess from 2:23 to 2:33.)

EXAMINATION

BY MR. LAGEMAN:

Q I'm looking here at what I believe is Page 11 of what we've been calling the notes. It describes Plaintiff's work with a product identified as cable hole filler compound.

A Yes, that's a subsection of the Occupational and Environmental History notes.

Q Okay. I just want to talk just briefly about this. I represent Lucent and AT&T in this matter. I'm having a little bit of difficulty understanding the actual notes section at the bottom.

Would you mind just explaining to me the significance of this -- of this section, what you are actually stating here?

A Sure. I guess I will just read it and explain as I go. "Identified asbestos exposure during mixing Western Electric asbestos filled bags cable hole filler media." What I'm really referring to is the discovery document from Western Electric that describes that media as asbestos-containing at least until 1974, and that's reviewed in another group of notes for the discovery documents. But that's what I'm referring to.

And then it says --

Q Memorandum of record, is that what we're talking about

here?
A That's correct, yes.
Q Okay.
A And then it says at the bottom, "Cutting/Ball peen hammer of the cover compressed gasket." And what I'm referring to is Mr. Quirin using the ball peen hammer to cut a hole cover gasket between the cover and the cable vault. And I refer to that as being described as a metal cover, and then I note that it's fire resistant.
And on the left-hand column, I note that the Lucent discovery describes as fire-resistant application consistent with asbestos. And I'm referring, again, to the discovery documents where they talked about a material being used between the cover and the cable vault as being asbestos-containing.
Q Okay. Well, let's just take them one by one. I just want to first talk about the cable -- well, what the plaintiff refers to as cable hole filler compound.
A Okay.
Q Correct me if I'm wrong -- well, why don't you just tell me what the basis is for your conclusion that that specific product, which he identified as being exposed to, contained asbestos?
A Well, the basis is the discovery document from Western Electric, the Memorandum of Record. It's indicated in

response to a March, 1974, memo that the Western Electric had asbestos-filled bags used as temporary cable hole filler media.
So my assessment of that is that that material would likely be asbestos containing at least until 1974 when the memo discusses phasing it out or cancelling it.
Q So what you are stating is that the R-9440 asbestos-filled bags which are referred to in that Memorandum of Record are in fact what is identified as cable hole filler compound in your notes; is that correct?
A Yes. In terms of Mr. Quirin's description of using it, it would fit with that material or that type of material.
Q Okay. So based on his description and the description of the asbestos-filled bags, you made a determination that those two products are in fact one and the same?
A Well, I haven't concluded that it's specifically R-9440, but certainly it describes the application of that as being asbestos-containing material. And certainly in the Lucent Technologies discovery document, they talk about the importance of it being a fire-resistant material.
Q Okay. The actual filler compound, we're not -- I'm staying away from the gaskets. So we'll talk about that in a second.
A Okay.

Q Are there any other grounds for your conclusion that the cable hole filler compound as identified by plaintiff is -- contains asbestos other than that it has a similar description to the asbestos-filled bags?
A No, it really is that description of it as a cable hole filler medium that is the basis for the opinion.
Q Okay. Could you -- do you have any experience or knowledge of how these asbestos-filled bags are used to fill these cable holes?
A Well, my determination is really from Mr. Quirin's description. It's not from any other use of that material.
Q Okay. So because you determined that they are the same product, and your description would match up with how Mr. Quirin described using it?
A Well, yes. I mean, they are basically describing a very specific application, cable vaults. And not only cable vaults, but the action of putting a cable through a hole in the cable vault. That's a pretty specific activity.
And the description of Mr. Quirin of mixing that and applying it certainly would indicate that that's the type of material being used, at least that's my determination to a reasonable degree of medical certainty.
Q So is it correct that you believe that the asbestos-filled bags contain the material that Mr. Quirin

describes as mixing to form the sort of sealant that was used to cover these holes?
A That would be my assessment.
Q And, again, that's based solely on the descriptions that you read in Mr. Quirin's testimony and in speaking with him and in this Memorandum For Record that you were provided?
A That's correct. And, yes, certainly he described that. And I guess I would add to that just my discussion with him in the interview just reviewing his occupational history.
Q Sure, sure.
Now, in another document that I have here discussing the document -- that is summarizing the documents you were provided, you refer to the asbestos cable hole bags R-440 as being called blue pillows; is that correct?
A There is a description that is in the Lucent Technologies discovery material of asbestos cable hole bags, yes, that are described as blue pillows by one individual. I believe in the interrogatories, they note that that is a term used for kale wool. They describe asbestos cable hole bags that were used between the cable vault covers for fire insulation protection, but they seem to distinguish that between the blue pillows.
Q Okay. I just want to move on briefly then to these

gaskets that you have discussed. I don't have -- I'm not familiar with the documents that you referenced, but I believe you stated that it was with regards to a metal cable vault cover that was coated with a heat-resistant material; is that correct?

A Well, that description is from Mr. Quirin's deposition. He did indicate he used a metal cable vault cover, at least at some point.

Q Okay. Now, how does the gasket -- if you could just explain to me conceptually where this heat resistant gasket would be located at least based on your understanding of what you have reviewed.

A Based on Mr. Quirin's description, it would be cutting a gasket that would fit between the hole and the cover. So it would be a gasket between those two materials.

Q And are we talking about a vault cover or a manhole cover?

A Well, my interpretation would be a hole cover in terms of use of that gasket.

Q Are you aware of where the hole -- I mean, would it be a hole, say, between a cable vault and outside or a hole from an underground duct to a cable vault?

A Well, again, the cable vault isn't something I have independently investigated, so it's really Mr. Quirin's description of that. It was a cover material. It seemed

consistent with the outside, but I don't want to over interpret that. It was his description.

Q Okay. So the cover then rested on what was described as -- or what was believed to be an asbestos-containing gasket?

A Well, Mr. Quirin didn't describe it as asbestos-containing. But certainly the description of the need for a heat resistant/fire resistant material for that application and the use of asbestos cable hole bags would be consistent with it being an asbestos-containing gasket during that period.

Q Are there other heat -- to the best of your knowledge, are there other heat-resistant materials that could have been just as easily used?

A Well, during that period of the 1960s in terms of heat-resistant gaskets, that would be typical asbestos containing. I mean, that would be the most likely. I mean, I can't -- and certainly Lucent Technologies describes material in that application as being asbestos. So that would be my conclusion.

Q Now, do you have any independent documents that discuss this or reviewed any independent documents that discuss this gasket or are you basing your opinions solely on the testimony of Mr. Quirin and the fact that these gaskets were described as being heat resistant?

A No, it is Mr. Quirin's description, as well as the discovery descriptions.

Q Okay. And, again, which specific discovery documents referred to a heat-resistant gasket?

A That would be in my file labeled Lucent Technologies. It's an interrogatory. I believe it's in the Taylor case.

Q Okay.

A It's been made Exhibit 9.

Q Okay. I apologize. I don't have the exhibits with me right now. Was that a Lucent or a Western Electric document or was that just a response to an interrogatory?

A I believe it was a Lucent response to an interrogatory. We're going to try to pull it out so we can be more specific about it.

Q Thank you. I would appreciate that.

A (Peruses documents.) It's Defendant Lucent Technology, Inc.'s Responses to Plaintiff's Supplemental Interrogatories and Requests For Production in Taylor v. Bondex, Harris County, Texas. And the date is March 6th, 2007, on that.

Q Does that have -- strike that.

Hello?

A Yes.

Q Okay. So it's a written response then? There's no Bates

stamped document?

A That's correct.

Q Okay. And the exact language of the response just references a heat-resistant gasket?

A Well, this interrogatory talks about a lot of different -- well, various asbestos-containing cables and thermostats that in my opinion are not applicable to Mr. Quirin's case, and I have not identified them as exposures.

But in terms of the asbestos cable hole bags, it does talk about a fire-resistant application. That's on Page 13 of the document. And it indicates that it was heat resistant. It was designed as a cable vault cover for fire insulation protection and could contain asbestos.

Q And that is the basis for your opinion that it was properly identified as an asbestos-containing material?

A Right. I mean, they call it asbestos cable hole bags, R-440.

Q Well, we're not talking about the bags. We're talking about the gaskets.

A No, I understand. But --

Q Okay.

A -- certainly this indicates a hot fire-resistant application.

Q Well, I guess I just want to make sure we're differentiating between the two. I mean, the asbestos-containing bags do use the word "asbestos." Is the word "asbestos" used with regard to these gaskets in any way?
A No. Mr. Quirin did not identify them as asbestos. He didn't know what the content was.
Q And did this discovery response that you were reviewing reference asbestos specifically with regards to this gasket?
A No.
Q Would it be safe to say that the primary basis of your opinion that it contained asbestos is because it uses the word "heat resistant"?
A Fire heat-resistant application, yes. That's the basis that it's likely asbestos.
MR. LAGEMAN: Okay. I have no further questions. Thank you very much.
THE WITNESS: Thank you.
MR. HALL: This is Eric Hall. I can go.
THE WITNESS: Good afternoon.
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EXAMINATION
BY MR. HALL:
Q Can you hear me okay?
A Yes. You are a little soft, but I can hear you.
Q Okay. I will try to speak up a bit.
Earlier there was a discussion about chrysotile as a causation for mesothelioma, and it's your opinion that chrysotile can cause mesothelioma, correct?
A Correct.
Q But you would agree that that's not a universally held opinion, wouldn't you?
A I would say it's a fairly large consensus opinion, and we've already cited IARC. But there are other organizations in addition to the broader World Health Organization, the societies of epidemiology, EPA, OSHA, NIOSH, ATSDR, and others.
There certainly is a discussion and debate about the relative potency of fibers. But I would say while some hold the opinion that chrysotile may not cause mesothelioma, I would say that opinion would be unusual. It would be certainly in the minority.
Q But you certainly have seen the opinions either in scientific literature or by specific scientists; isn't that correct?
A Well, the opinion I usually see is that it would require

a very high dose of chrysotile, some have that opinion, to cause mesothelioma.
The opinion beyond that that it can't cause mesothelioma period, I would say is a much more unusual opinion. Some may have that opinion, but I wouldn't call that part of the consensus. I think that is a more extreme opinion.
Q Are you aware of any fiber burden analysis in this case?
A I am not. In looking at Mr. Quirin's radical pleurectomy pathology, I did not -- I did not see that there was any evidence that there was lung tissue to assess fiber burden. So I don't believe there is.
Q Have you seen your -- the expert witness disclosure in this case?
A No.
Q Oh, okay. Well, there's some parts I want to ask you about, and that may go very fast. It says that you have expertise and training in biostatistics, epidemiology, toxicology and industrial hygiene.
I wanted to know if you have any actual -- any certifications in either of those -- any of those, biostatistics, epidemiology, toxicology or industrial hygiene?
A My certification would be within -- my board certification in occupational and environmental medicine

and my master's in public health and environmental health, that requires a demonstration of training and expertise in terms of biostatistics, epidemiology, toxicology and industrial hygiene, as well as occupational and environmental medicine, but it's within the field of occupational and environmental medicine.
Q In other words -- yeah, in other words, it's part of your training as a doctor, but, for instance, you are not a certified industrial hygienist?
A I'm not a certified industrial hygienist, but it's really a little more specific than a doctor certification because in occupational and environmental medicine, to become board certified in that specialty, it does require significant coursework as well as four months of on-site practicum interacting with industrial hygiene. So it's more rigorous than physicians in other fields would receive in terms of training.
Q Okay. I understand.
It has in your disclosure that you may testify that defendants knew or should have known that their asbestos-containing products or the use of asbestos-containing products could cause disease.
I'm here for Ingersoll-Rand. Do you have any opinions relating to Ingersoll-Rand's knowledge of asbestos-related hazards?

A Well, to clarify, my opinions regarding state-of-the-art knowledge really relate to medical knowledge as it evolved over time. I am not a corporate investigator. I do not investigate what companies knew and when they knew it. So I really cannot speak to that.

Now, I may be provided interrogatories that give me information about an entity's knowledge, and I can speak to whether that would be commensurate with medical knowledge at the time, and I could address hypotheticals like that as well. But I haven't undertaken for any entity in this case to investigate what they knew and when they knew it, and that would be true for Ingersoll-Rand.

Q Okay. And so far the information that you have for Ingersoll-Rand is contained in Exhibit 14; is that right?

A Yes, that's correct.

Q Okay. And have you seen any dose reconstruction information for any of the exposures in this case?

A As I indicated in a prior response, I have not performed a fiber cc year cumulative calculation. That's not part of my practice of occupational medicine, and I have not done that for any specific product or seen reports regarding that.

Q There's a portion in here that says that Dr. Brodkin will testify as to the following specific issues, and one of

those issues is ethical aspects of hazard communication, warnings and the state of the art for the medical and scientific literature regarding asbestos.

Do you have any specific training relating to warnings and their sufficiency as far as, say, governmental regulations?

A No. I would say my assessment of warnings is really part of the occupational and environmental history to assess whether hazard communication did occur and if it did, did it impact an individual worker's behavior in a way that would affect exposure. So it's really through the occupational and environmental history.

While I have written about ethics in occupational medicine, I would say that's fairly broad in terms of obligations to report hazards. But in terms of the hazard communication process, it's not through the regulatory process that I have any expertise because I'm not a regulatory expert.

Q And I'm guessing that you don't have any opinions at this point as to whether any company in this case ever failed to comply with any regulations?

A The word "fail" to me implies a legal liability question. I'm not a legal expert to address that. I certainly can address from an occupational medicine perspective what should happen in terms of controlling exposures and

reducing disease risks, but I cannot speak from it in terms of regulatory requirements or liability.

MR. HALL: Okay. Those are all the questions I have for you. Thanks.

THE WITNESS: Thank you.

MR. MILOTT: Doctor, this is Steve Milott. Can you hear me?

THE WITNESS: Yes. Thank you.

EXAMINATION

BY MR. MILOTT:

Q Doctor, how long was your conversation with Mr. Quirin?

A I've indicated the times on my notes. I believe it was 40 minutes.

Q And you had a single conversation for 40 minutes with Mr. Quirin; is that correct?

A Correct.

Q And when was that conversation?

A It was December 12th of this year.

Q As far as you know, that conversation occurred after you were hired by his lawyers and after he filed this lawsuit; is that right?

A That would be my understanding.

Q What is the job of an actuarial? Do you know?

A Actuarial evaluations are part of a mathematical

statistical analysis of various populations in terms of what predictions can be made in terms of demographic parameters. In terms of the health parameters, life expectancy is one of the parameters that's looked at. Certainly I rely on that in terms of the Social Security Administration life tables.

Q Are you done?

A Yes.

Q Okay. Do you know what education or training an actuary has in order to become a professional in that area of expertise?

A Well, I can't speak to the specific educational requirements, but it would require a firm foundation in mathematics and statistics.

Q Do you have a firm foundation in mathematics and statistics in order to become an actuary?

A I am not an actuary. I have not performed any actuarial analysis. I have relied on an actuarial analysis done by the Social Security Administration in terms of their life tables, but I am not an actuary.

Q And you don't have any education in that field, correct?

A No, I do not consider myself an actuary.

Q Am I correct?

A You are correct.

Q And you don't have any specialized training in that

field; is that correct?
A That's true.
Q You told us that you believe that amosite asbestos is about three times more carcinogenic than chrysotile; is that correct?
A I didn't say that. I said in terms of mesothelioma, it's three times more potent than chrysotile. In terms of lung cancer, there would be no potency difference.
Q And in terms of -- and thank you for clarifying that, and I apologize for asking my question the way I did.
You know that there are other experts in molecular biology, for example, who opine that amosite is about 100 times more potent a carcinogen than chrysotile in the analysis of mesothelioma; isn't that correct?
A I'm aware that there are opinions that there are greater potency differences in terms of amosite and chrysotile. I'm not sure which analysis you are referring to in terms of a 100 times. I know Hodgson and Darnton in Applied Occupational Hygiene did make that estimate of difference.
I would note that that has been heavily criticized in terms of their reliance on exposure information that is likely not specific enough to make that particular conclusion. That's been heavily criticized by Rodgers and others. But some do have that opinion.

Q And do you believe that those who hold the opinion that amosite is about 100 times more potent for purposes of mesothelioma, a carcinogen over chrysotile, are wrong; is that correct?
A It's my opinion that the evidence doesn't support that difference in relative potency. And then there are opinions on the other side. Certainly Nicholson and Smith and Wright have made opinions that -- or reached opinions that there's no potency difference between amosite and chrysotile in terms of mesothelioma.
I disagree with that as well. I think there is some relative potency difference. But it would not be in the order of a 100 times in my opinion based on the totality of the evidence.
Q Well, let me ask you this: If at trial the plaintiff's counsel was to put another one of their experts on the stand to say that the potency difference is about 100 times more in the case of amosite over chrysotile, you would disagree with that expert in terms of a mesothelioma finding, correct?
A Yes, I have a different conclusion. It would make no difference if they were plaintiffs or defense expert.
Q And you know that amosite asbestos was the primary type of thermal insulation used and installed on World War II era warships, correct?

A I am aware that amosite was one of the fiber types used in insulation aboard Navy vessels. I wouldn't conclude that it's the dominant one. I haven't done an inventory of insulation fiber types aboard naval vessels. It's not really my area. But certainly amosite was used in that application.
Q You would defer to naval experts as to how much amosite or chrysotile was used in the application of thermal insulation aboard World War II era warships, correct?
A Well, certainly someone who had inventoried them. There may be vessel to vessel variation as well. It would have to be someone knowledgeable about that as well.
Q Well, you know that amosite insulation existed in tonnages aboard World War II era warships, correct?
A Well, I'm aware that insulation could be measured in tonnages aboard vessels, whether it's amosite or chrysotile. Again, it's really outside my area. I have not assessed tonnages aboard the naval vessels.
Q So you just don't know what the tonnage might have been of amosite asbestos on Mr. Quirin's ship; is that correct?
A That's true. I have not investigated that.
Q Do you know that amosite asbestos was the primary source of thermal insulation applied to the steam lines on World War II era warships though; isn't that correct?

A Well, my answer would be the same. I'm certainly aware that it likely would have been a component of the material used for insulation in steam applications. Again, I can't speak to whether it had a primary role. It may have in some applications, it may not in others.
Q Do you know whether amosite asbestos was used on cold lines on World War II era warships?
A I'm not specifically aware of that. It's not to say that it wasn't. But certainly in terms of the medical industrial hygiene literature, my conclusion would be it likely would be involved in hot applications. It's not to say it wouldn't be used in cold applications, but I wouldn't assume it was.
Q You just don't know; isn't that correct?
A Well, again, I haven't investigated specific applications, but I would not reach a conclusion that a cold application would be asbestos containing.
Q So you don't know whether amosite was used on cold lines on World War II era warships, correct?
A Well, again, it may have in particular applications, but I wouldn't make a generalization that amosite was used on cold lines. I don't see a basis for that.
Q I'm asking you if you have knowledge or not whether or not amosite was used on cold lines on World War II era warships. Do you know?

A In terms of cold lines, I am aware that asbestos could be used, as well as non-asbestos.
Q I said amosite.
A As well as non-asbestos materials. I can't speak specifically to amosite.
Q Okay. Well, let me go on.
You know that Mr. Quirin was on a World War II era warship for about three and a half years, correct?
A Yes, the USS Tolovana.
Q And you know that he lived on that ship, that he worked on that ship, he ate on that ship for all those three and a half years, correct?
A Yes, except for the times he may have been off duty, yes.
Q You don't know too many of those times where he was off duty, though, do you, during that three and a half years?
A Well, his description was that they were fairly active in those three years.
Q When you say "active," you mean underway, right?
A Correct.
Q And you know that one of his main jobs was to maintain and repair and replace steam piping aboard that ship, correct?
A That was one of his jobs. Often in conjunction with equipment work, he would work on piping as well.
Q You do know that there were miles of pipes throughout

that ship, correct?
A Again, I can't speak to the distance, but I am aware that on naval vessels, there could be miles of piping. He did describe the Tolovana as a 500-foot oiler, so it was a fairly large vessel. So it seems likely there could be.
Q You know that he used a knife and a hammer and even his hands to tear off the old insulation from steam lines throughout his three and a half years on that ship, correct?
A He did describe a knife and I believe a use of a saw as well.
Q Do you remember him saying he pulled it off with his hands sometimes?
A Yes, he could pull it off with his hands.
Q And you know that those actions performed by Mr. Quirin would create dust in his breathing area, correct?
A Yes.
Q You know that he cut new pipe insulation for use on those same steam lines, correct?
A Yes, at times he would replace it.
Q And you know "at times" was throughout his three and a half years on that ship, correct?
A Yes, it would be during that three-year period.
Q You do know that he mixed mud insulation from a powdered form of asbestos to apply onto those lines; is that

right?
A That's true. He did that.
Q And you know that those activities caused dust to rise up into his breathing area, correct?
A Yes, that's true.
Q And after that dust would settle, it would be swept up and would rise up again in his breathing area all over again; isn't that right?
A There certainly is the potential for re-entrainment on a vessel like that.
Q As a matter of fact, you do recall his testimony saying that happened, don't you?
A I believe they did some of their own cleanup work as well, yes.
Q And you know that Mr. Quirin reported seeing dust below the main deck during his watches, correct?
A Yes, at times he did.
Q So I suppose what I'm getting at is whether or not all that information you were able to glean from reading his deposition transcripts and talking to him for 40 minutes that one time informed you as to whether or not his exposure to amosite asbestos for three and a half years was causative of his mesothelioma?
A In my opinion, certainly amosite exposure along with chrysotile in the Navy would be a component part of his

cumulative exposure that resulted in mesothelioma. It would certainly be a substantial contributing factor in his development of mesothelioma in my opinion. I've certainly indicated that in my diagnostic conclusions in terms of insulation aboard the naval vessel.
Q Well, would you agree with me that Mr. Quirin's exposure to amosite asbestos while he was working in enclosed areas such as the galley or the shops or the laundry room or the engine room, that would be more potent an exposure for causation purposes than if he was exposed to chrysotile asbestos while on the outside decks?
A Well, first of all, I don't agree with the characterization that he would have been exposed in the galley. He was working mainly with spigots in the galley. He didn't describe pipe work in the galley. He didn't describe -- he never did pipe work in the engine room. So I would disagree with that characterization.
But, yes, in the laundry room, he did replace piping that likely would have represented mixed fiber exposures in an interior compartment that would have been greater than outdoors, and that would be true for the pump rooms as well. He would certainly at times remove insulation from piping and flanges to access equipment, and that would be within interior compartments. So, yes, those would be sources of exposure.

Q They would be greater sources of exposure than sources of exposure -- for the purposes of a diagnosis of mesothelioma, they would be more potent and greater sources of exposure than to chrysotile asbestos from gaskets out in the main decks outside; isn't that right?
A I've identified insulation as one component of Mr. Quirin's exposure. I would not necessarily say they were dominant compared to other sources. He worked with insulation, but he worked significantly with gaskets and packing.
One of his primary work responsibilities was to maintain equipment, pumps and valves, and working with gaskets and packing was integral to that activity. So those are important exposures along with the insulation.
In terms of levels of exposure, Mr. Quirin described uses of chisels, hand wire brushing, scraping that certainly have been documented by Longo and others to cause exposures up to about 24 fibers per cc for hand applications.
That number is actually fairly consistent or similar in magnitude to some of the levels described at the high end by Balzer and Cooper for removal of insulation in terms of isolated insulation removal.
The packing would be somewhat less, but certainly up to four fibers per cc with removal as a significant

exposure.
So these are all component sources of his exposure. I don't make a list of priority in terms of which one was more important. I think they are all significant components of Mr. Quirin's cumulative exposure.
Q Well, those levels of gasket and packing exposure that you just talked about, those were found to have occurred in enclosed areas, and I'm talking about the four walls, a floor and a ceiling, right?
A Yes, those would be done in some indoor or protected context.
Q None of them were done outside, right?
A No, those studies weren't done outside.
Q Do you know that Mr. Quirin specifically testified, and I will quote, that "Most of the valves that I worked on were outside on the deck"? You know that, right?
A Well, that would be true for the winch pumps. That wouldn't be true for the pump rooms.
Q I'm talking about the valves. Mr. Quirin specifically testified, and again I will quote, "Most of the pumps I worked on were outside on the deck," close quote. Do you recall that testimony?
A Well, he certainly did describe 12 winch systems that used pumps and valves. So that's something he did. But he also did that work in the pump rooms.

Q Can a single fiber of amosite asbestos cause mesothelioma in a human being?
A In my opinion no. A single fiber of any of the major commercially available asbestos would not be a cause of mesothelioma. First of all, the body has significant respiratory defenses that would make that extremely unlikely. Those defenses have to be overcome by very significant exposures.
And, secondly, studies, pathologic studies, of individuals in the general population have certainly indicated a body burden of asbestos associated with individuals breathing ambient air, and there's no evidence that those individuals are at increased risk for mesothelioma either.
So I don't -- I don't feel a single fiber theory is scientifically valid.
Q Okay. You know that Mr. Quirin couldn't identify the manufacturer of any gasket or packing that he may have used in conjunction with the valve, correct?
A I think that's fair. He didn't know the brands that he removed. I don't think he provided testimony about the sheet gaskets that he utilized to replace them. He did indicate that at times he used manufacturers' gaskets but didn't provide a name.
Q Well, based on your professional expertise, your own

experience in studies you have done, you've learned that gaskets and packing used in conjunction with valve applications, if they contained asbestos at all, they contained chrysotile asbestos, correct?
A Yes, typically gaskets and packing for hot applications during the period that Mr. Quirin worked would represent a chrysotile application unless it were some sort of chemically resistant application, which could be mixed fiber.
Q Do you know there are some experts, medical professionals, some expert pathologists, who opine that only an amphibole asbestos fiber such as amosite, as Mr. Quirin was exposed to on that ship, can be the cause of mesothelioma, correct?
A Well, I think we've reviewed that. I've given you my opinion, and I think that's part of the consensus. I mean, as I said, some may have the opinion that chrysotile cannot cause mesothelioma. I think that's an outlier in terms of opinions.
Q One second, please, Doctor.
Do you know there are expert medical professionals and pathologists who will opine that chrysotile fibers such as might be in gaskets or packing that might have been used in the valves that Mr. Quirin worked on cannot be a cause of mesothelioma, correct?

A Well, there may be those people with that opinion. But I think one has to characterize what is done with the gaskets and packing. For example, if you look at the textbook by Selikoff and Lee, they talk about gaskets not being an important cause of disease in the form used in equipment. But in terms of the method and activity of using it, if it disrupts the material, it's a significant risk factor for disease. So it takes a consideration of that.

Again, I think if airborne fibers are being generated on a significant basis, chrysotile fibers would be a risk for disease. I think most professionals in my field would have that opinion. I'm not saying everyone does.

Q By the way, Doctor, your report, your notes on Page 5, if you can turn to that, please?

A Okay.

Q Somewhere on that page, I don't have it in front of me, but I know it's on Page 5, your notes cite that Mr. Quirin worked on hundreds of valves. Do you see that?

A Correct. That's in the occupational and environmental history subsection.

Q And you wrote that regarding his work on valves while he was on that naval ship, correct?

A Correct.

Q There's no work on valves subsequent to his time in the Navy, correct?

A Correct.

Q Now, that cite that you have about him working on hundreds of valves, I have got to tell you I deposed Mr. Quirin, and I know that he said he saw hundreds of valves aboard that ship, but can you cite me to a page and line from anywhere in his testimony where he says he worked on hundreds of valves on that ship?

A Well, these are notes I took from reviewing the deposition. I mean, the deposition is going to speak for itself. I mean, if you want me on a break to look for it, I might or might not be able to find it. I mean, I would be glad to look for it.

Q That's okay. You will defer, as far as correctness as to how many valves he may have worked on, you will defer to the transcript of the deposition itself; am I correct?

A Yes. But in terms of the totality of it, I mean, one has to be careful about taking one line out of context. I mean, this was my assessment based on the totality of it. But, yes, the deposition will speak for itself.

Q That was your assessment based on the totality of what, of reading the deposition transcript?

A Yes. This section of notes is based on my review of Mr. Quirin's deposition testimony.

Q And somehow in reading that transcript, you came up with the fact that he worked on hundreds of valves; is that what you are saying?

A Yes, I've written it down. Yes.

Q You don't know how many valves in fact he worked on while he was on that ship; is that correct?

A Well, Mr. Quirin's testimony was not to the resolution of a specific number of valves.

Q Am I correct?

A Yes, so you would be correct in that.

Q And you don't know how many times he may have changed out a gasket or packing from a valve; isn't that correct?

A Not in terms of a specific number, but it was a regular practice that he performed in terms of his machinist duties.

Q Am I correct, Doctor?

A You are correct in terms of a specific number.

Q Okay. You know that there were machinists and other naval personnel who worked on the valves aboard that ship other than Mr. Quirin, correct?

A That's true.

MR. MILOTT: Doctor, I have nothing further. Thank you.

THE WITNESS: Thank you.

MR. COOK: Hello, sir. This is Eric Cook. Can you hear me all right?

THE WITNESS: Good afternoon. Yes, thank you.

EXAMINATION

BY MR. COOK:

Q Doctor, I represent Union Carbide. And I have -- I'm going to jump around quite a bit because we have covered some ground already. So if you need some more context for my questions, please let me know.

A Thank you.

Q You had mentioned at the beginning different folders that you had for a number of different defendants for the case. Do you have a folder specific for Union Carbide in this case?

A No.

Q Do you have any information that Calidria was incorporated into any of the products that Mr. Quirin worked with or around, sir?

A Some of the discovery documents for the joint compound do discuss suppliers. And give me one second. (Peruses documents.)

In the Georgia-Pacific discovery document, they do refer to one of the suppliers as being Union Carbide,

which I would typically associate with Calidria asbestos.
Q And in that one, does that refer to two other suppliers of asbestos as well for Georgia-Pacific?
A Correct.
Q And with specific regard to the joint compound that was used while Mr. Quirin was present, do you know whether or not Union Carbide Calidria was incorporated into that joint compound as compared to the other two suppliers?
A No, I'm not a supply expert that researches supply lines.
Q Do you have any information on Mr. Quirin's dose from any alleged exposure to Calidria in this case, sir?
A No. Again, let me just ask for some clarification about what you mean by dose. Are you talking about fiber cc year?
Q Well, you had mentioned earlier in response to some questions that dose was an important consideration for you, and so do you have any information as to dose, whether it's fibers per cc year or another way of determining dose, sir?
A Well, in my field of occupational and environmental medicine, I rely on the occupational history. And certainly that history would indicate in terms of this use of chrysotile asbestos that Mr. Quirin would have had regular bystander exposure over a 20-year period between approximately 1957 and 1977 in the joint compound.

Now, in terms of Georgia-Pacific, he indicated that he saw that in the latter part of his work perhaps as a supervisor, sort of the '67 to '77 timeframe that would be relevant. But that's a qualitative assessment. It's a duration of exposure that could include approximately a 10-year period.
It would be significant intensity exposure in terms of mixing, sanding and sweeping joint compound that Mr. Quirin was in proximity to, but I can't speak to the specific fiber type supplier. It was chrysotile in my opinion. I can't speak to who the supplier was.
Q All right. And so if I understand your answer correctly, sir, with specific respect to Calidria chrysotile, you have no information as to Mr. Quirin's alleged dose; is that correct?
A That's true. I have not done a supply specific assessment. I certainly did note that UCC was one of the suppliers. I mean, it certainly informs my opinion that it was a chrysotile exposure. But it doesn't tell me what dose or how much by supplier.
Q All right. And in that vein, sir, that presents a possibility that Calidria was incorporated into the joint compound that was used while Mr. Quirin was present, but it doesn't rise to the level to give you enough information to provide an opinion to a reasonable degree

of medical certainty as to Calidria, does it?
A Again, I cannot provide testimony about who the suppliers are to that specific joint compound. I believe, I'm just thinking about the other joint compound Mr. Quirin saw and cites US Gypsum. I mean, I haven't researched it or looked at any other interrogatories in this case, but I believe Union Carbide was one of the suppliers for that entity as well.
But, again, I'm not a supply expert. I can't really speak to whether the material Mr. Quirin worked around contained a specific supplier, whether it be Union Carbide or someone else.
Q Okay. I'm going to move topics on you, sir. And I've read some of your previous testimony where you testified that the Verma and Crump final draft on potency estimates was rejected by the EPA. Do you recall that testimony?
A Well, they created an analysis where coefficients established potency differences. That recommendation was not adopted by the EPA. I've testified to that.
Q And you would agree that not being adopted by the EPA is different than being rejected by the EPA, correct?
A Well, certainly it was an analysis that was done for the EPA for consideration. Based on that consideration, it was rejected for adoption. Now, I guess it's semantics is what you would want to call that.

Q And is there a particular document that you are referring to that specifically says it was rejected, sir?
A No. I believe I'm aware of a letter of correspondence, I don't have it with me, so I don't want to cite it specifically, but it certainly speaks to not adopting it.
Q All right. And is that the letter from Cane with regard to the Bratton proposal of 2008?
A Yes, I believe the letter was authored by Cane.
Q All right. And it specifically referred to the Bratton proposal, correct?
A Well, I don't have it in front of me, so I don't want to really speak to specifics of it. But it was a letter by Cane speaking to the use of that selective methodology between the fiber types.
Q Are you familiar with the Bratton 2008 proposal, sir?
A Well, without it in front of me, I don't want to get into specifics. So, you know, I don't have a working memory of the specifics of it.
Q Okay. And so you would defer to the contents of the letter as to whether or not it rejected Bratton, 2008, as compared to Verma and Crump, 2003, correct?
A Yeah. And, I mean, I'm not sure we're even talking about the same letter. It may be the same author and not the same letter. So I don't know.
Q Do you know when the letter you are referring to was

authored?
A I don't want to guess. Sitting here, I just don't want to be inaccurate about that.
Q Okay. Let me change topics on you again then, sir. Let me ask you briefly just some basic questions about animal studies. What are the limitations of animal injection studies for determining disease causation in humans?
A Well, there can be certainly biologic variability between species. So while toxicologic assessments of animals can be extremely useful in assessing biologic effects, it shouldn't be considered definitive in assessing what would happen in terms of human experience.
So interspecies variation is a very important limitation. The other limitations would be dose response for some animal experiments. Significant doses are provided that may result in a different physiologic response than might be experienced in typical human experience or inhalation. So those are limitations.
Q In addition, an injection study bypasses the body's defense mechanisms as well, correct?
A It does, and it generates physical injury potentially by the application mechanism that may also be a confounder and would have to be controlled for.
Q All right. You would agree, sir, that there are substances that cause mesothelioma when injected into

animals but do not cause mesothelioma when humans are exposed to those substances, correct?
A Yes. I mean, there are good examples of different injection experiments that just by virtue of injuring the tissues cause mesotheliomas that wouldn't be applicable to causation of mesothelioma in humans.
Q Sir, Calidria has never caused mesothelioma in an animal inhalation study, has it?
A The studies I'm aware of, and I've cited a number of them in the reference reliance list I brought for Calidria, which is a NIOSH pure chrysotile, so it's been used in a number of animal experiments, have been injection studies or there are some studies that have utilized cells in culture, including human mesothelial cells in culture.
I'm not aware of a specific inhalation study. I mean, certainly there are inhalation studies of chrysotile, but I don't recall them being specific to Calidria.
Q All right. You, in reaching your opinions with respect to Calidria, have not reviewed any inhalation studies then?
A No. I mean, I really don't distinguish Calidria from other forms of chrysotile. I mean, for example, Coffin and Cook studied chrysotile relative to amphiboles and found high rates of mesothelioma, but that wasn't

specific to Calidria. I wouldn't consider it a Calidria experiment, but it does inform my opinion about chrysotile.
Q You would agree there are important physical differences -- strike that.
You would agree there are important differences in the physical characteristics of Calidria as compared to other forms of chrysotile though, correct?
A Well, again, I'm not a mineralogist to really speak to those aspects. In my opinion as a physician in occupational medicine, Calidria would be a form of pure chrysotile exposure that would have the biologic and health effects of chrysotile. It is certified by NIOSH as a pure chrysotile, so it would be substantially free of contaminants, and I would consider it a pure form of chrysotile. But I don't consider it a mineral distinct from chrysotile.
Q All right. But going back to my question, though, and I just want to make sure I understand your answer, sir, because you are not a mineralogist, you have not considered any of the physical characteristics that differentiate Calidria chrysotile as compared to other forms of chrysotile?
A In terms of the mineralogic physical aspects, no, with the exception that Calidria is a relatively pure

chrysotile that would distinguish it from some other deposits.
Q I'm sorry, sir. I'm flipping through stuff that we have already covered.
Sir, you have Kanarek, 2011, listed in your -- the reference list at the end of your notes.
A Kanarek, yes.
Q Yes.
And my question for you on that is what flaws or limitations of Kanarek, 2011, did you consider in reaching your opinions in this case?
A Well, it's a review that informs my opinion. It reviews numerous articles that are -- the original articles are published elsewhere. So I don't use Kanarek independently of looking at the original epidemiologic studies, but I think it's a useful review of those studies. I certainly have relied on it in that context.
It's not original research in terms of health outcomes associated with asbestos exposure, but it does, I think, provide a cogent review of many of those studies.
Q All right. Are there studies that are omitted from Kanarek that you believe should have been included in his review?
A Well, I don't have it in front of me. I mean, I think

it's a useful review of the literature. I don't know that it's a comprehensive review of all the literature. But rarely is a review that.

So without having it in front of me, it's hard for me to address that specific question. I mean, I think it was a well done review in my opinion in terms of reviewing important studies. That's not to say it reviewed all studies.

Q Okay. I'm going to change topics on you again, sir, and ask you just a couple of state-of-the-art questions.

Do you have an opinion as to when it was generally understood in the United States that mesothelioma was a primary malignant tumor of the pleura?

A Well, I think that understanding developed in terms of -- well, let me ask for clarification.

Are you talking about an asbestos-related mesothelioma?

Q Well, I'm going to actually ask you just first with respect to mesothelioma, regardless of whether or not it's connected to asbestos.

A Mesothelioma was first characterized, to my understanding, in the 1930s, but it wasn't initially appreciated that it could be an asbestos-related phenomenon.

Q All right. And -- but you would agree that there was

debate in the United States as to whether or not mesothelioma was a primary malignant tumor of the pleura prior to the 1960s, correct?

A I would say it was established as a primary pleural malignancy in the 1930s. But, you know, certainly knowledge evolved over time. I mean, it's a rare tumor, and there definitely was discussion of whether that could represent secondary malignancies. It took time to certainly establish it was a primary pleural malignancy.

Q When you say it was established, sir, by that what do you mean?

A That it was a tumor -- a malignant tumor of origin in the mesothelial tissue of the pleura.

Q Okay. And I guess what I'm getting at, though, is when you say established, is that the first report saying that mesothelioma was a primary malignancy of the tumor or -- because my question was addressed to when it was generally understood.

A Right. And, again, I mean, you might want to talk to a pathologist about that, that specific question. It certainly is discussed in the medical literature as a primary pleural malignancy independent of a pulmonary malignancy even in the 1940s.

If you look at the 1947 case report of malignant mesothelioma, that's discussed as a primary malignant

mesothelioma.

Now, there is some discussion in there about whether it could be from some other source, but they rule it out.

Q Okay. So -- and I guess once again my question wasn't about whether there was discussion about it, but when it was generally understood, sir.

So with respect to when it was generally understood that mesothelioma was a primary malignant tumor of the pleura, you would defer to a pathologist; is that correct?

A Well, no, I would refer -- defer to a pathologist in terms of a discussion of the pathologic findings. But in terms of a discussion of mesothelioma as a primary pleural tumor, I mean, I cited the New England Journal of Medicine, I would also cite JAMA in 1949 that distinguishes lung cancers from pleural cancers. I think that was established in the 1940s. I mean, that wasn't an area of ongoing debate.

Now in discussions, there would always be an assessment about whether or not a tumor of the pleura could have metastasized from some other primary site. I mean, that's part of a differential diagnosis. But in terms of there existing a primary pleural tumor, no, I think that would have been established in the 1940s and really in the 1930s.

Q All right. And let me just read to you from another article that you cited in your reference list here, sir. And this is from Wagner, 1960, Diffuse Pleural Mesothelioma in Asbestos Exposure in Northwestern Cape Province.

Beginning with the discussion in that, it says specifically, and I understand you don't have this in front of you, sir, but I will represent to you this is what it says, "In 1924, Robertson denied the existence of primary malignant tumors of the Pleura and considered them to be secondary in origin. Since then, on the one hand, Willis 1948-1953, and Smart and Henson in 1957 have supported Robertson's views, while on the other hand primary neoplasms of this nature have been described by many authors in recent years."

And so Wagner, 1960, what he's indicating there is there was actually a debate as to whether or not mesothelioma was a primary malignant tumor of the pleura through that time period, correct?

A Well, I think it's referencing the discussion that pleural malignancies can commonly be metastatic from other sites. So it's part of the discussion.

I mean, I think Wagner's discussion is fair that, you know, by that timeframe, it was pretty well established, but it was still a source of discussion

among some individuals. And that's because the pleura can be a source of metastases.
Q Sir, do you have an opinion as to when it was generally agreed in the United States that asbestos could cause mesothelioma?
A Again, there's not a bright line date in any of these medical questions. It's evolution of knowledge over time.
I would say that first citation I gave you in terms of the New England Journal of Medicine, 1947, would be the first established case where an individual exposed to asbestos cutting insulation board developed a documented pleural mesothelioma.
But from that point, there were other cases in series, certainly Vice reported mesothelioma in a valve repair worker in marine settings and reviewed Wedler's finding of 31 individuals with asbestosis of which two developed mesothelioma.
And then certainly Cartier in 1952 describing mesothelioma in chrysotile-exposed miners, as well as Lecker describing peritoneal mesothelioma in the 50s really led up to Wagner's paper, which I think more definitively established asbestos as a cause of mesothelioma.
Q I'm going to change topics on you again, sir.

Do you have an opinion as to the relative contribution of early exposures as compared to subsequent exposures of ten or more years later?
A Well, I think if there are exposures of ten or more years latency, I would consider them all relevant assuming it's an identified exposure from the occupational history that, you know, it would meet the criteria of a significant airborne asbestos exposure that would overcome the body's burden and -- overcome the body's defenses and add to the body's burden of asbestos that would increase risk for mesothelioma.
I don't really have a methodology to weigh one exposure over another based on latency as long as it meets that 10-year latency criteria. I know Peto and others have tried to model waiting for earlier exposures, but I think most of the risk really comes from dose, not latency.
Q Okay. And when you refer to Peto, you are referring to Peto's statement that the risk of asbestos is actually determined from the time since first exposure; is that correct?
A Correct.
Q All right. Have you read the more recent articles on that?
A There have been other publications about it.

Q Have you reviewed and considered those in reaching your opinions in this case?
A I have reviewed some. I don't know which ones you are considering. But, yeah, there have been several articles about that. I really consider dose within an appropriate latency. I don't rule in or rule out exposures just because there's a certain latency unless it doesn't meet some minimum latency criteria.
Q Let me ask you this question: If the only exposures that Mr. Quirin had were the exposures to asbestos-containing insulation that he identified during his naval service, and then he subsequently developed mesothelioma and was diagnosed on the same date that he was diagnosed in this case, would it be your opinion that those exposures to asbestos-containing thermal insulation were sufficient by themselves to cause his mesothelioma?
A Well, I would indicate that I guess I would treat it as a hypothetical question. It's obviously not Mr. Quirin. Certainly one can be exposed to pipe insulation. It increases risk for mesothelioma and one can develop mesothelioma.
An individual that had Mr. Quirin's exposure to pipe insulation would be at increased risk for developing mesothelioma. That individual wouldn't be at as great of risk as Mr. Quirin was by virtue of his other exposures

to gaskets, packing, drywall, insulation in the telephone land-based setting and others. But it is an exposure that would increase risk for mesothelioma, and such an individual could develop mesothelioma.
Obviously in that assessment, I would want to assess their comprehensive occupational and environmental history.
Q All right. Let's say you have a hypothetical person with three and a half years of exposure to amosite-containing thermal insulation. In your opinion is that sufficient to cause pleural mesothelioma?
A Well, I certainly would identify that as an exposure, and it could increase risk for mesothelioma in a particular individual. It might be the only exposure that one identifies.
But certainly, again, one would want to take a comprehensive occupational history because mesothelioma is a cumulative dose disease. You have to consider all the sources of exposure.
Q My question is, sir, if the only exposure that was presented was three and a half years of exposure to amosite-containing thermal insulation, would that exposure be sufficient in your opinion to cause pleural mesothelioma?
A It could, yes. I mean, clearly that exposure would

increase risk for mesothelioma and such an individual could develop mesothelioma, absolutely. But, again, one wants to consider the comprehensive occupational history.
Q Let me switch topics on you again, sir, and let me talk to you just briefly about hazards and risks.
Would you agree that there's a distinction between a hazard and a risk?
A Yes.
Q Would you agree with the definition of a cancer hazard as an agent that is capable of causing cancer under some circumstances?
A Yes. I mean, I would say a hazard would occur when there is a known carcinogen with a potential route for exposure. That would result in a hazard should an individual become exposed.
Obviously it doesn't define a hazard. It's a potential hazard depending on the circumstances.
Q Right. And so you cannot agree with the definition of a cancer hazard as an agent that is capable of causing cancer under some circumstances unless you qualify your answer then?
A You would have to qualify it. I mean, just because a material is toxic doesn't mean that it's hazardous.
Q Would you agree with the definition of a cancer risk as an estimate of the carcinogenic effect expected from

exposure to a cancer hazard?
A Repeat the question. I'm not sure I understood it.
Q Of course.
Would you agree with the definition of a cancer risk as an estimate of the carcinogenic effect expected from exposure to a cancer hazard?
A Well, yes. I mean, risk is the resulting effect of exposure, so I think as a basic principle that's true. And certainly that addresses dose response.
Q You would also agree that the distinction between a hazard and a risk is an important one, correct?
A Certainly.
Q Sir, I think that is all -- oh, I'm sorry. I forgot one area.
You had mentioned Balangero, and that's the chrysotile cohort in Italy, correct?
A Correct.
Q Now, you are familiar with balangeroite?
A I am, yes.
Q And balangeroite is a contaminant of the chrysotile at the Balangero mine, correct?
A It's a mineral that exists in that same strata.
Q All right. And actually has been likened to tremolite as a contaminant of chrysotile, correct?
A Well, it's been discussed. I think in terms of studies,

there's not been an identified health effect of balangeroite. But that has been discussed in terms of the mineralogy.
Q You also mentioned Yano and the chrysotile in China, correct?
A Yes.
Q And looking at that subsequent studies have demonstrated that there actually is significant tremolite contamination in the chrysotile mines in China, correct?
A There's been some discussion of tremolite contamination. I mean, often in these areas of relatively pure chrysotile, there are discussions of the mineralogy, whether there's some level of contamination.
I think the conclusion is that the Chungking province has relatively pure chrysotile. That's not to say there aren't some contaminants. I am aware that that's been discussed, although, again, I don't really focus on the mineralogic literature.
Q Well, focusing specifically on fiber burden, fiber burdens of individuals in the region that were reportedly exposed to pure chrysotile have actually demonstrated very high levels of tremolite in the lung tissue, correct?
A I would want to see the article you are referring to. I don't want to make a blanket statement about that.

Q Okay. And so you have not reviewed any articles indicating an elevated level of tremolite in lung fiber burdens of individuals in the region of the China chrysotile mine in reaching your opinions in this case?
A Well, again, I don't have Yano's articles in front of me to speak to that. There may have been some discussion of whether they saw some amphibole fiber. But in terms of it being a major contaminant, I'm not aware of that. So --
Q I'm sorry. Were you finished?
A Yeah. So, I mean, if you have a specific article, I mean, I would have to -- I would have to look at it. I just can't refer to it as I sit here.
Q In your opinion, sir, what level constitutes a major contaminant?
A Well, again, I'm working in the dark here a little bit because I'm not sure what article you are referring to, whether it was part of the lung burden, if they saw one isolated tremolite fiber in a lung digestion analysis, or whether we're talking about percentages, you know, within a strata of chrysotile. So I'm not sure what you are referring to.
Q Well, I'm asking you -- sir, you specifically said a major contaminant. And so I'm curious in your mind and in your opinion what level of tremolite would constitute

a major contaminant. And you can tell me in any form that you like, whether it's a fiber burden or as indicated from the mine.

A Well, I'm talking about some isolated finding of amphibole within the general context of the exposure. I mean, it's difficult in a place like Chungking because you are talking about chrysotile that doesn't have a lot of biopersistence in the lung. We've talked about that at length earlier today.

So it can be very difficult to assess chrysotile in terms of body burden which biases towards finding amphibole. So those are issues in the lung burden studies.

In terms of just mineralogic studies, I mean, it can vary. I mean, tremolite in Libby, vermiculite can be well over a percentage of the entire mineral. I mean, I would call that a very significant exposure, a very significant contaminant.

If you are talking about contaminants that are, you know, orders of magnitude below one percent, they could represent trace exposures.

Q If the -- if the lung fiber burden indicated tremolite above background, would you consider that a major contaminant?

A Not necessarily, particularly in a chrysotile lung burden

study where it tends to skew the results towards finding amphiboles. I mean, I think such a study would really have to compare it to other referent ranges. And, again, without a study in front of me, it's really hard to speak to it.

Q And in your reference list in this case, you haven't cited any studies addressing that issue, have you?

A No.

MR. COOK: All right, sir. I think that's all the questions I have. Thank you very much.

THE WITNESS: Thank you.

MR. SHOR: Hi, Dr. Brodkin. Can you hear me okay?

THE WITNESS: Yes. Good afternoon.

EXAMINATION

BY MR. SHOR:

Q My name is Eric Shor, and I represent Hollingsworth & Vose Company. And I want to turn to your -- the section of your notes entitled Exposure-Related Documents Reviewed and Considered. Can you pull those out?

A (Peruses documents.) Okay.

Q And this is a four-page fax, and I want to see if you can turn to Page 3 of that fax.

And at the top of that page, it says,

"Exposure-Related Documents (Cont'd), P. Lorillard Co. Cont'd." And then it says "Industrial hygiene evaluation of H&V Specialties Co." Do you see that?

A Yes.

Q And you say here, "Operator exposure of 3.5 to 11 million particles per cubic foot," right?

A Yes.

Q And you are aware that H&V Specialties was told that the maximum allowable concentration for that facility was 15 million particles per cubic foot?

A As I sit here, I really only recall what I have written in the notes, that there was concern about it being above the TLV of 5 million particles per cubic foot. That's not to say there wasn't a discussion of 15. I just don't recall it as I sit here.

Q You don't remember that it said in the document that you reviewed that because asbestos was less than a third of the material that was being used in that plant, they tripled the 5 million and used 15 million particles per cubic foot as the maximum allowable concentration for that specific facility?

A I think there was some discussion that it wasn't pure asbestos, that there was cotton and cellulose and other materials. But, again, I don't have the document in front of me right now.

Q But in fact you are not aware of any evidence that H&V Specialties was told that their plant exceeded the maximum allowable concentration, right?

A No, I'm not aware of any sort of violation or a document like that, no.

Q And you mention further down that there was a concern that 5 million particles per cubic foot of asbestos dust is too high.

You are aware that 5 million particles per cubic foot was the current threshold limit value in 1952, right?

A Yes, that's correct.

Q And when was that changed?

A That was changed in 1968 down to 2 million particles per cubic foot. That would be the date of transition.

Q And after you say "concern that 5 million particles per cubic foot asbestos dust is too high," you have a parenthetical that says "lower levels required for illness prevention" and then a bracket, "Dr. Elkins 11/3/1952," right?

A Correct.

Q And you are referring when you say that to a memo from Dr. Elkins to someone named Mr. Whalen, right?

A It is a memo from Dr. Elkins that I was referring to when I took these notes.

Q And it was not a memo from Dr. Elkins to H&V Specialties Company, was it?
A Boy, the document would speak for itself. I mean, I have no reason to question what you are saying. I just don't have it in front of me.
Q There's no evidence, for example, that H&V Specialties Company was ever told that officials were concerned that the five million particles per cubic foot level was too high and could cause illness, right?
A I can only speak to the memo, itself. I can't really speak to the line of communication within H&V.
Q But Dr. Elkins didn't work for H&V, did he?
A You know, I would have to look. These are notes I took a while back, so I'm not sure who his employer was. I really can't speak to that.
Q Do you know -- do you know who Dr. Elkins is?
A I don't specifically. I mean, I haven't researched it.
Q Well, if the memo that you are referring to said "no copy of the report is being sent to us by the employer," would that inform your opinion about whether H&V Specialties was ever informed of this specific memo or its contents?
A I would have no knowledge of the communication line. I mean, if there's a memo that says they weren't informed, that would speak for itself.
Q So to the extent you have this information in your notes,

you are not using this information to say that, for example, H&V Specialties was aware that exposures in its plant were above a safe level, right?
A Well, I think this evaluation speaks to certainly a health concern based on release of asbestos fibers. I mean, it informs my opinion that the filter media material is capable of releasing asbestos fibers and at least in this context was capable of releasing them to a level that generated health concerns.
Q But this was not the filter media releasing anything; this was the filter media being manufactured, right?
A That's true, yes.
Q So this is before it was ever put into rolls, right?
A It was the carting, processing of the material.
Q So then this material is carted and processed and put into rolls, right?
A Correct.
Q And then it was shipped to Lorillard in another state, right?
A That's my understanding, yes.
Q And then after that in another state, Lorillard used that material to make cigarette filters, right?
A That is my understanding.
Q And then those cigarette filters were attached to cigarettes?

A Correct.
Q And then they were put in packs?
A Correct.
Q And then those packs were put into cartons?
A That would be my understanding.
Q And then they were shipped off to different places where people purchased them, right?
A Correct.
Q So what happened at this plant in Massachusetts doesn't tell you whether the filter media, when a Kent cigarette was smoked, released asbestos, does it?
A No, I don't think this evaluation speaks to that. But I think it speaks to the assessment of workers fabricating that material that they can be exposed to high levels that would generate health concerns.
Q And you say toward the end of these notes, there's a recommendation for CXR, which means chest x-rays, for exposed workers, right?
A Correct.
Q And there's no mention in the documents you reviewed of any safety measures that should be implemented for individuals who smoked Kent cigarettes, is there?
A No, I don't recall seeing that.
MR. SHOR: All right, Dr. Brodkin. That's all I have. Thank you for your time.

THE WITNESS: Thank you.
MS. RAINES: All right. I guess it's my turn.
Does anyone need a break? Doctor?
THE WITNESS: We can take a five-minute break, sure.
(Recess from 4:02 to 4:09.)
(Exhibit No. 27-28 marked for identification.)

EXAMINATION
BY MR. SCHILLING:
Q Dr. Brodkin, my name is Drew Schilling. I represent several defendants in this case.
Earlier you had mentioned you created folders for various defendants in the asbestos litigation. And I just want to go over to see if you've created folders for any of my specific clients. All right?
First is Warren Pumps, do you have -- have you collected any information in order to form any kind of opinions regarding products manufactured by Warren Pumps?
A No. I didn't pull any files regarding Warren Pumps in this case, so I haven't brought any in. I don't believe it was mentioned in Mr. Quirin's deposition.
Q So at this time -- well, the materials you reviewed in

preparation for your deposition here today did not have anything to do with Warren Pumps specifically, correct?
A No. I did not assess Warren Pumps. Certainly I assessed pumps in general, but not anything that Mr. Quirin identified specifically as Warren Pumps.
Q The second defendant I want to ask you about that I'm here for is Parker Hannefin. Do you have a folder created for Parker Hannefin?
A No, not in this case.
Q Do you have Parker Hannefin materials from previous cases?
A I might, but I certainly didn't consult them or consider them in this case, so I didn't pull them.
Q And so at this time you don't have any specific opinion in regards to Parker Hannefin, itself, in regards to the Quirin case?
A No. I mean, I don't know what hypotheticals I might be asked at trial regarding those entities, but they wouldn't be independent of my opinions about the exposures I've identified.
Q And would this be true for Imo Industries as well?
A Yes.
Q Which would be Delaval as well, correct?
A Correct.
Q And I'm going to guess you don't have any materials in

regard to Molex, Incorporated, correct?
A That's true.
Q And you haven't reviewed any materials specific to Molex, Incorporated, in preparation for your deposition here today, correct?
A Only in terms of Mr. Quirin's deposition, not any other documents.
MR. SCHILLING: I said I would be brief, and those are all the questions I have. I appreciate your time.
THE WITNESS: Thank you.
MS. RAINES: I'm Elizabeth Raines. We met many hours ago.
THE WITNESS: Yes.

EXAMINATION
BY MS. RAINES:
Q Good afternoon, Dr. Brodkin. I'm here representing Lorillard Tobacco Company. I would like to start with the documents you have brought with you that are contained in two Redwelds I have sitting here on the table.
I believe earlier you said these are your Kent micronite materials from the Burns and McGuire cases that inform your opinions; is that right?

A That's true.
Q And did you actually review the contents of these two Redwelds for the Quirin case to inform your opinions?
A I certainly perused them. I didn't specifically look at every single document, but I did peruse them.
Q I would like to go ahead and just identify what is here for our record in this case.
As Exhibit 28, we have marked the Redweld, and it says -- oh, I'm sorry. We're on 27. Let's start over. All right. Strike that Exhibit 28 reference.
Let's start with 27. And we have the Redweld marked Burns, Charles, and that signifies the Charles Burns case, correct?
A Correct, discovery documents I received in that case.
Q And you served as an expert witness for the plaintiff in that case?
A That's correct, yes.
Q And are there any documents in this Redweld that you collected yourself versus receiving them from a plaintiff's lawyer?
A I would have received those in the context of a medical/legal action through a lawyer.
Q All right. And inside this Redweld are five folders, so I just wanted to --
(Interruption in proceedings.)

MS. RAINES: Wow. Are you okay on the phone?
(No response.)

MS. RAINES: All right. We'll go on.
Q (By Ms. Raines) We have five folders inside the Redweld. The first one has been marked as Exhibit 27A. And on it, it says Kent Micronite/P. Lorillard documents. Correct?
A Yes.
Q And inside that folder is a CD labeled Lorillard documents and then a stack of documents with various Post-it notes on it, correct?
A Yes.
Q These notes that are stuck on the documents in the folder we've now marked as 27A, are those all from the Charles Burns case or are there new notes for the Quirin case?
A No, I have not added any new Post-its in the Quirin case. They would have all existed before.
Q And these are -- actually the CD in here is labeled Kananian Plaintiff Trial Exhibits Re Lorillard Tobacco, correct?
A Yes. Mr. Kananian was an individual I evaluated a number of years ago. And I believe I incorporated the discovery documents I received in that case in the Burns case.
Q So all of that is contained in 27A?

A Correct.
Q Moving on to 27B, you have a folder labeled Kent Micronite/Hollingsworth & Vose, correct?
A Correct.
Q And in this folder are discovery responses from the Robert Cox case; is that right?
A Yes, another individual I evaluated in the context of Kent micronite.
Q So you served as an expert witness for the plaintiff in the Robert Cox case?
A That's true.
Q And the notes that are attached to the discovery responses from the Cox case that are contained in Exhibit 27B, are those previous or do some of those correspond to the Quirin case?
A They are previous. I have not added any Post-its for the Quirin case.
Q Next, 27C, says Kent Micronite Discovery Documents on it, correct?
A Yes.
Q And inside this folder are a number of paper documents and a DVD labeled Fullam, I believe, TEM series Kent articles; is that correct?
A Yes.
Q And then there are a number of paper documents and some

photographs of Kent cigarette packs and advertising, correct?
A Correct.
Q And there are also a few Post-it notes here. Were all those placed by you previously or are any of those new for the Quirin case?
A They were all placed previously.
Q 27D is labeled Dr. Longo testimony 8/18/95, Micronite v. Raybestos, correct?
A Yes.
Q And inside this folder it appears are two transcripts of testimony from the Horowitz case; is that right?
A That's my understanding, yes.
Q And, again, there are a number of Post-it notes with handwritten notes on them. Are any of these handwritten notes on the Post-it notes new for the Quirin case or are they all previous to this case?
A No, they would all be previous.
Q And then finally Exhibit 27E is the folder labeled Trial Testimony of Douglas Hallgren, Horowitz versus Raybestos, correct?
A Yes.
Q And, again, this folder also contains a transcript from the Horowitz versus Raybestos case with a number of Post-it notes with handwritten notes on it, true?

A True.
Q And are any of these Post-it notes new for the Quirin case?
A No.
Q They are all previous to this case?
A They are all previous.
Q All right. That completes Exhibit 27 and 27A through E.
Moving on to Exhibit 28, we have a Redweld labeled McGuire, William, correct?
A Yes.
Q And that was from the William McGuire case in Kentucky in which you served as an expert for the plaintiff; is that right?
A Correct.
Q And we have Exhibit 28A, which is a file folder labeled Lorillard Cases Death Certificates, McGuire, William, true?
A True.
Q And did you review this again for the Quirin case?
A No. I perused it but certainly did not add any Post-its. I reviewed the findings from my prior review.
Q Next is Exhibit 28B, which is labeled Trial Testimony of Douglas Hallgren, Cox versus Asbestos Corp, McGuire, William; is that correct?
A (Peruses documents.)

Q It's hard to see.
A Yes, that's a transcript.
Q The folder only contains the transcript which also has a number of Post-it notes with handwritten notes on it, correct?
A Correct.
Q And are any of those notes new for the Quirin case?
A No.
Q And then finally Exhibit 28C is labeled Owens-Corning Testimony of Kent Micronite/Testimony of Mark Risler, R-I-S-L-E-R, Ph.D. (additional material), and that is abbreviated, McGuire, William.
A Yeah, that's probably --
Q Is that right?
A It's probably Rigler.
Q Oh, Rigler. I know that name.
A Yeah. It's testimony. I believe I probably received this after my deposition in the McGuire case.
Q And so this is testimony you reviewed after your deposition in the McGuire case, but before the trial in the McGuire case; is that right?
A That's my recollection.
Q And did you testify at the trial in the McGuire case?
A No.
Q So you simply reviewed this testimony, and have you ever

testified in a case involving the Kent micronite cigarettes that contained asbestos since the McGuire case?
A No, not to my recollection.
Q All right.
A That would be the most recent one.
Q And so in these notes you've put on Post-it notes on Exhibit 28C are from the period of the McGuire case and not for the Quirin case, correct?
A Correct.
Q Now, earlier your comprehensive set of handwritten notes was marked as Exhibit 18. And that includes the list of various publications on various subjects related to asbestos exposure. What section of your notes refers specifically to publications that inform your opinions about the Kent cigarette in this case?
A The publications that inform my opinion relevant to Kent would be on the final page. It's a list of articles that would start with Dodson and Hammar, Inhalation Toxicology, 2006, and end in Millette, MVA 2010.
Q And earlier I believe you testified this would be a cumulative list of reliance documents; is that right?
A That's correct. As new articles come to my attention or I review them, I periodically do add them to the list.
Q And are there any publications that you've added to this

list since you gave your expert opinions in the McGuire case?
A Yes. The bottom two, I did receive these reports in the context of the Quirin case and have added them.
Q And one of those is Rigler and Longo, MAS 2012, correct?
A That's correct.
Q And why was that one added to your list?
A Well, I certainly reviewed it. It was among the materials sent to me in this case. And it certainly is an analysis of release of crocidolite fibers during the process of smoking from a smoking machine.
So I certainly found it relevant to understanding exposure levels relative to smoking with the asbestos-containing Kent micronite filter.
Q And did that publication change in any way the opinions you expressed about the Kent asbestos-containing filter cigarette from the McGuire case?
A I would say it doesn't change any of my opinions from the McGuire case. It does provide some additional information. This analysis looked at fiber cc ranges or intensity of exposure, whereas previous analyses that Longo had done had looked more in terms of structures. And the technique was different. Rather than using a piston method of smoking, they used a standard smoking machine. So it's additional information that I included.

Q So is it correct to say that your review of this Rigler and Longo MAS 2012 report did not cause you to form any new opinions about the Kent asbestos-containing filter cigarette?
A I think that's true. I think it refined some of my opinions about levels of exposure just based on use of the standardized smoking machine.
Q Did it cause you to actually try to calculate an exposure level for Mr. Quirin in this case?
A No, although, the report, which I don't know what exhibit -- maybe it would be Exhibit 7, does look at an individual who smoked six Kent cigarettes per day. So that actually is in the range of Mr. Quirin.
So that does inform my opinions as well about the type of cumulative exposure that might result. It's in the appendix of that report. I mean, I certainly read and considered it.
Q When you say "the appendix," are you referring to Table 2 in the MAS report that's been marked as part of Exhibit 7 in this case?
A Actually, what I'm referring to is dose of crocidolite structures calculation for Kent micronite cigarettes for six smoked per day for seven days. So there is that analysis that I certainly looked at and considered.
Q That were smoked on a smoking machine, correct?

A Correct, yes.
Q And these were 60-plus-year-old Kent cigarettes that were smoked on a smoking machine, right?
A Yes, they would have been vintage from the '52 to '56 timeframe.
Q And you don't know how those Kent cigarettes that Longo tested which resulted in this report, you don't know how those were stored for all those 60 plus years, correct?
A I can't speak to that. My only knowledge of it would be the description in the report and the methods they used. But, no, I wouldn't have personal information about that.
Q And you can't speak to the validity or the reliability of the testing that is reflected in this July, 2012, report by Rigler and Longo, correct?
A I would say I certainly would rely on the material from the scientists in terms of measuring the exposure levels and the methodology. I mean, I'm not a smoking machine expert. I don't have expertise in that technical aspect of it. So I certainly rely on their expertise and measurements. They certainly describe it in the report.
Q So this isn't actually a test that is representative of a person smoking a fresh Kent cigarette between 1952 and 1956 when there was asbestos in the filter, true?
A The smoking machine was smoking a cigarette from that era with the asbestos-containing chrysotile filter. It was

treated in the standard method for smoking machines such as described in the report. But, yes, it would be representative of that cigarette.

Q But you are not a material scientist, correct?

A Correct. I'm relying on the material scientist's analysis in terms of understanding the exposure levels.

Q But you can't say whether the materials tested, the Kent filter cigarettes that were tested, were actually representative of those at the time from a material science perspective?

A Again, I rely on Rigler and Longo for that.

Q Right. And you are not an expert in testing cigarettes for particle or fiber release, correct?

A Correct. I don't do that as part of my practice.

Q And you've never designed a cigarette filter, right?

A No.

Q And you've never designed any type of filter at all; isn't that right?

A That would be true.

Q Okay. While we're talking about testing documents that you reviewed, there's also the MVA scientific consultants expert report dated September 30th, 2010, and you've added that to your reliance list, correct?

A Correct.

Q And so how does this report inform your opinions in the

Quirin case?

A The Millette report speaks to a TEM or transmission electron microscopic analysis of the Kent micronite cigarette. It confirms release of crocidolite fibers in four of the filters tested. So certainly that informs my opinion about release of fibers from the filter.

It also indicates some technical limitations from the scanning electron microscope versus the transmission electron microscope in terms of their analysis. So I've noted that.

Q And, again, here we're analyzing 50- to 60-year-old Kent cigarettes, correct?

A Yes, vintage 1952 to 1956.

Q And, again, you don't know how those were stored or how any materials in the cigarette filter may have degraded before they were tested by MVA, correct?

A Again, I rely on the reports in terms of their representativeness. I don't have independent knowledge of it.

Q And actually in the MVA testing, they found that some of their Kent cigarette samples showed no release of crocidolite, true?

A They did, and certainly the scanning electron microscopy did not appear to show it. But what Dr. Millette notes is a likely limitation of SEM versus TEM.

Q And so the results there in the MVA test were four samples they say released some crocidolite fibers, but four other samples did not show release of crocidolite fibers, correct?

A They have noted the ones that showed release and those that didn't, and they speak to some of the limitations of the methodologies.

Q And in fact there was a broad range of results in terms of the number of fibers detected in the MVA testing, correct?

A There was variation, yes.

(Discussion off the record.)

Q (By Ms. Raines) Are there any other materials besides the Redwelds from the Burns and McGuire case, the Exhibit 7 with the two expert witness studies and your reliance list that's in your handwritten notes, is there anything else besides that that you are relying on for your opinions in this case?

A Just the ones specific to Mr. Quirin in terms of the occupational and environmental history.

Q In terms of his smoking history?

A Exactly.

Q Okay. Earlier you said you looked at plant exposures regarding Kent micronite cigarettes. Why did you do that

when Mr. Quirin was not a plant worker?

A You are referring to the West Groton plant, the earlier questions?

Q Yes.

A Well, it certainly is material that I reviewed earlier and had taken notes from. Again, it's a different application. It's an occupational setting where the media is being -- the filters are being fabricated. It's not to say that would be analogous to smoking the cigarette. But from a health standpoint, in occupational medicine one wants to understand the occupational exposures as well as the environmental exposures. So it is relevant that you understand the workers making the materials as well.

Q All right. But Mr. Quirin wasn't one of those workers, so that's not relevant to his exposure here, correct?

A I would say the exposures during carting of the media to create the filters is certainly not synonymous with smoking the Kent micronite filter. I don't use the report in that way.

Q All right. And you looked at materials from the Burns case, but you are not doing that to make a comparison between Mr. Quirin and Mr. Burns, correct?

A That's true. In fact, I've not reviewed the medical details of Mr. Burns for this case.

Q And the same question for McGuire, you are not reviewing materials for McGuire because you are going to make some sort of comparison between Mr. McGuire and Mr. Quirin in this case, correct?
A That's true.
Q And you are not going to give any opinions that any other individual who smoked Kent cigarettes is somehow similar to Mr. Quirin, correct?
A Well, I think one has to assess each individual in terms of the evidence. I mean, certainly I would rely on the literature I've cited and that we've gone over in terms of my understanding of the exposures related to Kent micronite and the effect of smoking Kent micronites on lung burden. And I would rely on those studies. But certainly I would consider Mr. Quirin as a specific individual in terms of his smoking history.
Q I believe you previously testified that you earned about $800,000 in 2011 and $400,000 of that was for medical/legal work; is that correct?
A That sounds about right for 2011 -- well, 2011, $850,000, somewhere in that range. That's my recollection.
Q All right. Would that make your medical/legal work income for 2011 $425,000?
A Yes, it would be about 50 percent of my activity in 2011. It's probably a little more this year.

Q And what is your estimate for your income this year in terms of medical/legal work?
A The amount would be roughly the same. I would say $850,000. But probably about 60 percent of my activities would involve that rather than 50.
Q Okay. So more along the lines of, you know, $500,000 plus earned in 2012 for medical/legal work?
A Yeah, I mean, I haven't figured out an exact number, but I think 60 percent of $850,000 would be roughly the range.
Q Okay. Have your opinions regarding relative potency of different fiber types in causing mesothelioma changed since your October 2011 deposition in the McGuire case?
A No.
Q Okay. And have your opinions regarding TLV threshold or asbestos exposure level required to cause mesothelioma changed since you were deposed in the McGuire case?
A No.
Q And have your opinions regarding ambient air levels, that they are trivial exposures changed since you were deposed in the McGuire case?
A No, they haven't changed, and certainly I would not attribute asbestos-related disease to ambient levels of asbestos exposure.
Q And have your opinions regarding state-of-the-art

knowledge of asbestos and mesothelioma changed since you were deposed in the McGuire case?
A No.
Q Have your opinions regarding possible causes of mesothelioma changed since you were deposed in the McGuire case?
A No.
Q And have your opinions that ship and shipyard workers' risk of asbestos-related disease is significant changed substantially since you were deposed in the McGuire case?
A No, and I think I certainly have provided those opinions today as well.
Q Have your opinions regarding the Kent filter composition when it had asbestos in it, manufacturing specifics and the patent information changed since you were deposed in the McGuire case?
A I would say in broad overview, those opinions haven't changed. I would note the Rigler and Longo MAS 2012 study that we talked about. I believe they cited that crocidolite was about 9.8 percent of the filter. They had not reported that on previous studies, so that's some new information.
It doesn't substantively change any of my opinions, but to the extent there's some new information in that report, I've considered it.

Q All right. But the Rigler and Longo report and the Millette report that we talked about earlier are both expert reports and experiments prepared for litigation, correct?
A That would be my -- well, certainly I received them in the context of this litigation. I mean, I guess I can't speak overall as to what the context would be unless they've indicated it in their reports. Those would speak for themselves. But I received it in the context of litigation.
Q Well, and I will hand you Exhibit 7, which has your copies of the Rigler/Longo and of the Millette reports, and you can tell me if you see anything in there indicating these are peer-reviewed published studies?
A It would be my conclusion that they are not peer reviewed. I mean, these are material science reports. There's no indication here that they are in a peer-reviewed publication. They are reports.
The MVA report is -- well, at least there's a cover letter to the law firm of Waters, Kraus & Paul, so it was submitted in the medical/legal context. I don't know if it was originally written in that. But in terms of the MAS report, they call it an expert report, so I would assume it's in some medical/legal context likely.
Q Are you aware of any peer-reviewed published

epidemiological studies of Kent micronite asbestos-containing filter smokers?

A I'm not aware of a study of smokers. I'm aware of the worker studies I've already spoken about. I'm aware of assessments of lung burden in individual smokers, Kent micronite smokers, asbestos-containing Kent micronite filters who developed mesothelioma. But I'm not aware of a study to date that has systematically looked at smokers during that era. I'm just not aware of that.

Q So your reliance list that we talked about earlier does not contain a single peer-reviewed published article that reports on an epidemiological study of Kent asbestos-containing micronite filter smokers, correct?

A I would say there isn't a population epidemiologic study of Kent micronite smokers that I'm aware of that's ever been done. I'm just not aware of that.

Q And would you agree with me that there's a hierarchy of reliability for medical and scientific evidence?

A Yes.

Q And probably at the top would be your clinical studies and then your lab studies and then epidemiological studies and then case reports, and then at the bottom would be studies and reports in the legal context, correct?

A Well, I'm not sure I would characterize it that way. I

think within epidemiology there is a hierarchy of evidence that would go from case reports to case series to population-based studies and design perspective studies.

In terms of other reports, they may inform opinions about exposure that really are independent of health effects. I mean, those are a different type of report and certainly could be material science reports that may be in the context of peer-reviewed literature or not.

But I would consider those and toxicological reports are an important complement to human studies, but I don't -- I mean, I would just consider them complementary, not a substitute for human epidemiologic studies, but certainly one that is a group of studies that should be considered in terms of causation.

Q But you would place a peer-reviewed published study above a study done in the context of litigation and not peer reviewed and published, you would place the peer-reviewed published study as more reliable, correct?

A Well, I think the peer-reviewed process establishes reliability and validity. So in that sense, yes.

Q Right. It would be more reliable and valid if it's peer reviewed and published than if it was just published for litigation?

A Well, I wouldn't say it would be necessarily more

reliable. I would say its reliability and validity would have gone through a peer-reviewed process and been established. That's not true for a report -- a study that hasn't gone through that process.

Q Have your opinions about the testing of the Kent asbestos-containing filter in the 1950s changed since you were deposed in the McGuire case?

A No, I've not received any additional information that would change my opinions regarding any of the Armour, the Fullam or Revere studies. No, those would be the same.

Q And have you reviewed those studies, the company testing, Fullam, Revere, Armour Research Foundation, for the Quirin case?

A Well, I perused them and certainly looked at my notes and brought them in in this case. So to that extent, yes.

Q And you didn't form any new opinions as a result of that review?

A Correct.

Q Have your opinions about Longo's 1990s testing of both published and unpublished results and the methodology changed since your deposition in the McGuire case?

A No.

Q And have your opinions about company knowledge of asbestos hazards in the 1950s changed since you were deposed in the McGuire case?

A No.

Q And you have not formed an opinion in which you quantify fiber release from Kent cigarettes for this case, correct?

A Just to clarify, in terms of a calculation of a fiber cc year, no, I have not done that for any entity in this case. It's not part of my practice.

Q And I wanted to make sure that fit into your earlier testimony about the other products.

A Right. My assessment is all from the occupational and environmental history. It's a qualitative assessment of duration and certainly understanding the intensity of the exposure through the various studies I have cited, but I haven't calculated the fiber cc year.

Q I'm just checking my notes to make sure I'm not repetitive.

You've testified in a few previous asbestos cases involving alleged Kent cigarette usage including Kamanian, McGuire, Cox, Burns and others, correct?

A Those are the major ones I can recall sitting here.

Q And in each of those cases, you were under oath to tell the truth just like you are today, true?

A Correct.

Q And do you reaffirm your testimony in each of those previous asbestos cases involving alleged Kent cigarette

use?
A Yes, I would.
Q Okay. And you previously testified extensively about your qualifications and areas of expertise including areas where you say you are not an expert. That has not changed since your previous testimony, correct?
A True.
Q Have you ever reviewed any opinions of any of the other experts who have been designated by the plaintiff to testify in this case?
A In terms of an actual assessment of Mr. Quirin?
Q Yes.
A Well, I can't really speak to the Longo 2012 report if that was done for the Quirin case or not. If it was, then I have. But I reviewed it whether it was independent of Mr. Quirin or not. And I'm not aware of any other report that would be specific to the Quirin case.
Q Have you had any conversations or meetings with any of the other expert witnesses that the Quirins have designated to testify for them in this case?
A I haven't had any meetings with any other experts in this case.
Q Have you had any meetings or conversations with any of Mr. Quirin's treating physicians?

A No.
Q Let's talk about Mr. Quirin's smoking history specifically. Now, Mr. Quirin smoked brands other than Kent, correct?
A That's true.
Q And his deposition testimony indicates that although he says he smoked Kent while he was in the Navy, he also smoked other brands when he was on leave in foreign ports, correct?
A Yes.
Q Because he says Kent wasn't available in foreign ports, right?
A Right. He purchased the Kent micronites from the ship store. If he wasn't aboard ship or they weren't available, he would get other cigarettes.
Q Did that impact your opinions in this case in any way?
A I would say not in terms of the general duration of exposure. Mr. Quirin indicated that shortly after he boarded the Tolovana, he started smoking the Kent micronites that were available in the ship store. And then at some point after he left in '57, he transitioned to another cigarette. In my opinion, that defines a period between mid 1954 and mid 1956 that he was likely exposed to the Kent micronite.
Now, could there have been some days he didn't smoke

it? Yes. But as I testified earlier, this was an active sailing vessel. For the most part, Mr. Quirin was at sea with pretty limited time away from the ship. So the majority of it certainly would be Kent micronite. It's not to say that every day was Kent micronite.
Q And how much did Mr. Quirin say he smoked per day while in the Navy?
A My assessment of that was approximately a third of a pack per day. It could range from five to seven cigarettes per day.
Q And how does that amount of smoking per day that Mr. Quirin has claimed affect your opinions in this case?
A In my opinion, it certainly defines a period of approximately two-thirds of a pack year specific to Kent micronite cigarettes. It is a significant cumulative dose. I have discussed that in the diagnosis and assessment section of my notes, and that would be on Page 4 of that subsection in terms of a third of a pack per day for two years.
And certainly based on the published Longo study of 1995, one can look at the estimated range of crocidolite structures for a third of a pack per day, and they do represent exposures in the millions of crocidolite structures. So in my view, that would be a significant exposure to crocidolite.

Q But you are not saying that was Mr. Quirin's dose of crocidolite from smoking Kent cigarettes as he's claimed he did, correct?
A Well, I don't think I can give a specific dose. And as I said, I haven't calculated a specific dose in this case. I think what these numbers tell me based on Dr. Longo's assessment, and I've also cited his 2012 numbers, is that there is an intensity of exposure in the 2012 analysis in the 4 to 68 fiber per cc range in the mainstream smoke that would represent part of his usual daily or almost daily exposure over a two-year period.
And certainly I would identify that as an exposure, a component part of the exposure, in the context of others.
Q And how did you come up with two years of exposure?
A Well, two years really relates to Mr. Quirin's testimony, his recollection, that he began smoking Kent micronites shortly after joining the Tolovana, that that was the cigarette he purchased from the ship's store. That really -- and if you look at the naval personnel records, that dates very specifically to May of 1954.
So in the general period of mid 1954, he started smoking Kent micronites. And although he stopped smoking them sometime after 1957, the relevant period really is to mid 1956 in terms of their asbestos content. So

that's where I get the two years, mid 1954 to mid 1956.
Q And Mr. Quirin didn't know for sure about the date when he started. He thought it was a few months after he came aboard the Tolovana, correct?
A Well, Mr. Quirin, and he indicated this to me in my interview, is not great at recalling specific dates. I mean, he just doesn't have a memory for that.
What he did have a memory for, though, is he started smoking those cigarettes shortly after joining the vessel, the Tolovana, and he was pretty consistent about that in his deposition testimony. I asked him about that, and I think he was consistent about that when I talked to him as well.
So I think that date is really pretty specific. It's not to the day or month, but I think it's pretty specific to mid 1954 for starting.
Q I just want to be sure I'm clear on the opinions you are planning to give at trial. Are you going to give an opinion about the amount of asbestos exposure that you think Mr. Quirin had from allegedly smoking Kent cigarettes from sometime in 1954 to 1956?
A I certainly would be comfortable providing the information that I provided in my notes at trial. I mean, I don't think there's going to be additional information. I guess the one piece of additional

information would be the 2012 Longo. That analysis indicated a somewhat lower dose than the 1995 analysis.
So I would -- I would opine that there's some range depending on the methodology used, the type of smoking machine. So I would provide that opinion that there's not one specific exposure, but I would be comfortable talking about those ranges in the two analyses that I considered.
Q Just the fact that that was reported or that you are saying that's the dose?
A Well, I'm not going to say that Mr. Quirin was exposed to this specific dose. I would indicate and I certainly would cite that 4 to 68 fibers per cc in the mainstream smoke as an intense exposure on a daily basis over that two-year period, and that analyses of cumulative smoking have indicated millions of crocidolite structures for an individual with that range of smoking, but not a specific level.
Q So are you going to pick some of these ranges or numbers as between Longo in the 90s, Longo in 2012 and Millette in 2010?
A I think it would be fairer to pick the 2012 lower range and the 1995 higher range as a range that's been observed and calculated that I've considered.
Q Why is that? I'm just --

A Well, it basically offers estimates of cumulative exposure or body burden of structures for that designated level of smoking. I mean, I have considered that in terms of assessing whether it was a significant exposure.
Q Okay. So looking at Longo's revised Table 2 that we talked about earlier, and I will give that back to you, that's Labeled Crocidolite Fibers in Eight Puffs Average, correct?
A Yes. Table 2 is labeled Actual Number of Crocidolite Fibers in Kent Cigarettes Smoked.
Q All right. Now --
A Oh, you are looking at the table at the end.
Q Yes. I'm sorry. Table 2. Because that's where the numbers are that we've been discussing, correct?
A Yeah, this one is called Table 2 as well. But, yes, I've looked at that as well.
Q There's a Table 2 and then a revised table?
A There seems to be an Appended Table 2 that breaks it into specific cigarettes.
Q Yes. And so there we have crocidolite fibers in puffs, which we've got 15,400 plus 8,120 plus 1,120, plus 19,040, plus 5,880. And I don't know if you added that up there in your notes, but I would represent to you that total is 49,560.
So if we want to figure out how many fibers per

cigarette, would we divide by five?
A That would give you the average, yeah.
Q Okay. And I will represent to you that when you divide 49,560 by 5, you get 9,912 fibers in cigarettes -- fiber per cigarette.
A That looks like probably it would be pretty close to the average, yes.
Q And then if we take 9,912 fibers per cigarette times seven cigarettes a day, a high number for Mr. Quirin's smoking, that equals 69,384 fibers per day; does that sound right?
A Yeah, I haven't done the math, but I have no reason to think it isn't correct.
Q So back in the 1950s, if you look at TLVs and what was known about asbestos exposure, is 69,384 fibers per day significant?
A Well, I don't think one can easily transpose this to TLVs. I mean, TLVs are a regulatory level for work-related exposures. It's sort of apples and oranges in terms of exposure. I mean, these are intense exposures that happen during inhalation of cigarette smoke. So I don't think I would make a comparison to TLVs.
Q But 69,384 fibers per day is not millions, correct?
A No. On a per-day basis, that's true.

Q It's not even close to millions. So when you talked about millions of fibers earlier, what time period were you speaking of?
A Well, for instance, Longo does an analysis for six cigarettes smoked per day here below Table 2, and he takes the average of fibers per cc and then does 35 cc's per puff times 8 puffs times 6 cigarettes and gets 59,000 crocidolite fibers per day. I'm not so sure that was different than the number you got. It was pretty similar.
Q Uh-huh.
A So that's the daily rate. You know, one certainly can calculate that over a two-year period as well.
Q So to get to millions, you would have to go not just days, but weeks?
A You would have to go to weeks. By certainly a month, you would be -- well, by three weeks you would be in the million range.
Q And that's assuming there was any fiber release from the Kent cigarettes that Mr. Quirin smoked, correct?
A Well, it would be based on the evidence that there is release here. I mean, otherwise one wouldn't make that conclusion. But these are the observations.
Q And there actually are some test results that you've reviewed for this case that show no measurable release of

asbestos fibers from Kent cigarettes when they had asbestos in them, correct?
A There is variation in the testing. So although Longo has reported a release in every cigarette, I don't know that one can say that for every study that has ever been done. But certainly in the 1950s studies, which you asked me about earlier, there was consistent demonstration of release in those fibers, too. So I think overall there is consistent evidence of fiber release.
Q But there are also some reports where there was no release or traces or three fibers in the case of Armour Research Foundation, correct?
A Right. Well, Armour -- I mean, the Armour one has to kind of assess in the context it was done. I mean, they were having difficulty with fiber overload with exhaled cigarette smoke -- or inhaled cigarette smoke. They designed a study to look at residual fibers during exhalation, which is going to greatly underestimate the fibers, and they did see several fibers even on exhalation.
Q And you would stand by your previous testimony about the Armour Research Foundation studies and the other Kent cigarette studies that were done in the 1950s, correct?
A Yes. My opinions haven't changed about them.
Q If Mr. Quirin did not smoke Kent cigarettes with the

asbestos-containing filter in the 1950s, what caused his mesothelioma?
A Well, I guess I would view that as a hypothetical. An individual who had Mr. Quirin's occupational history of asbestos exposure but did not smoke the Kent -- asbestos-containing Kent micronite cigarettes would also have had a significant cumulative exposure to asbestos that would place that individual at increased risk for mesothelioma and certainly could be causally associated with it.
Obviously an individual that had that exposure but had additional exposures such as Kent micronite or others hypothetically would be at further increased risk than that individual.
Q And if no fibers were released when Mr. Quirin smoked Kent cigarettes with an asbestos-containing filter in the 1950s, these cigarettes would not contribute to cause his mesothelioma, right?
A Again, I will treat that as a hypothetical because the evidence I reviewed would allow me to conclude otherwise. But in the hypothetical that there was no release of asbestos fibers, then it would not contribute to mesothelioma, it would not be a source of exposure.
Q Do you have an opinion on why Longo keeps getting results with fewer and fewer fibers released as the Kent

cigarettes he is testing age?
A Well, he discusses in the 2012 report the reasons for the variation between the various testing. He describes the difference as, first of all, using different methods and going from the piston method of inhaling to a standard ISO smoking machine. And then there are differences in the humidification of the cigarettes. I believe in the 1995 testing, he used a 90 percent humidification. The ISO method he used in 2012 required 60 percent humidification. That may account for the difference in fiber release. So there are those variables that he discusses.
Q But you are not personally familiar with the various ISO standards that apply to moisture content, storage of cigarettes, testing of cigarettes, any of those issues, correct?
A No, I certainly have relied on Dr. Longo and Dr. Rigler's discussion.
Q You are relying on what Longo and Rigler and Millette have stated in their reports, not on your own knowledge and study of filtration, filter materials or cigarette filters and testing of cigarettes, correct?
A Correct.
Q In fact, you can't say one way or the other whether any of these cigarettes that Longo has been testing are truly

representative of Kent cigarettes with the asbestos-containing micronite filter that were sold in the 1950s, true?

A I have no conclusions based on independent knowledge. Again, I do rely on the material science reports for that.

Q So if Longo or Rigler or anyone else is incorrect that those Kent cigarettes that have been tested are representative of what Mr. Quirin or anyone else claims they smoked in the 1950s, then those weren't representative, correct?

A Well, certainly Millette and Longo's report discuss the process by which they assess the cigarettes and why they feel they are representative.

I mean, in the hypothetical that they were not, then they wouldn't accurately reflect the nature of those cigarettes and potentially the exposures.

Q And then you wouldn't rely on those results to the extent you do today if that were true, correct?

A If I had evidence that they weren't representative, that would be true. But based on my review of the studies, they certainly address it and conclude that they likely are representative of the types of exposures that were experienced by individuals such as Mr. Quirin.

Q But if you had verification from a reliable source that

the Kent cigarettes tested by Longo and Millette are -- or were not representative of Kent cigarettes sold to consumers in the 1950s, then you would not rely on those test results, correct?

A In the hypothetical that they were not representative, I wouldn't rely on them.

Q Okay. And you have no method of isolating any particular exposure that Mr. Quirin may have had to asbestos in the context of disease causation, correct?

A Let me ask for some clarification on that question.

Are you talking about Kent micronites or the sort of extent of the other components of his exposure?

Q You can't take one alleged exposure and say that one caused his disease, correct?

A Absolutely not. Mesothelioma is a dose response disease. It's the aggregate dose that increases risk and causes disease.

Q So is it your opinion that Mr. Quirin's alleged Kent cigarette smoking in and of itself would not have been sufficient to cause his mesothelioma?

A I can't say that one way or the other. I don't think medical science has a way of addressing that question. Mr. Quirin's exposure to asbestos smoking the Kent micronite filters between 1954 and 1956 in my opinion is a significant exposure. I've identified it as a

component part of his exposure. That exposure is not as great as the cumulative exposure he had including all occupational exposures.

So the exposure to Kent micronite did increase his mesothelioma, but it's the aggregate of his exposure in the environmental as well as the occupational setting that resulted in his total risk and caused his mesothelioma.

So I have no way of parsing out a single component, whether it be Kent micronite or some other component.

Q You would agree that the smoking history you take is only as good as the information provided to you, correct?

A Yes. Certainly one relies on the history to assess cumulative smoking.

Q Do you have any other opinions you expect to give specifically concerning Kent filter cigarettes or Lorillard in this case that you haven't given in a previous case or we haven't discussed today?

A I would say not. My opinions are certainly provided in my notes, and I would reference those in aggregate. But as I said, I've not developed new opinions, and I think we've talked about the opinions I would express. That being said, I don't know what hypotheticals I will be asked. Certainly to the extent my knowledge, training and experience allows, I would address those.

Q So the only significance of your handwritten notes about the Lorillard documents is to show that you did review them again for purposes of your opinions in this case?

A Well, yes. While I'm certainly familiar with them, I did review them to refresh my memory and basically, you know, consider all the evidence I've looked at. So certainly they are part of the evidence I considered.

Q During the course of your review of Mr. Quirin's testimony or your interview with him, did you learn that his smoking was limited on the ship because it was a fueler, an oiler?

A Yes, they had designated smoking areas. Their smoking was limited.

Q And did he share the reason for that or did you learn the reason for that?

A I didn't discuss it with him in the interview, but he did discuss it in the deposition that because it was a fuel vessel, an oiler, he had to smoke in an area that wasn't necessarily the most pleasant area to hang out.

Q You noted in here that his smoking was limited. Did you note anything else about why it was limited or the rules on the ship that limited his smoking?

A No, I have no other additional information about that.

Q Did Mr. Quirin tell you that there were time periods when he wasn't permitted to smoke at all on the ship?

A Boy, the deposition would speak for itself about that. I don't have a specific recollection of that. It's not to say there wouldn't be.

I mean, I'm sure under certain watches, you wouldn't be allowed to smoke obviously. But in terms of not smoking period as a policy, I don't recall that.

Q He didn't tell you anything about that during your interview with him?

A No.

Q Do you plan to do any further work in this case before the trial?

A I feel comfortable with the materials I've been provided and considered in making my opinions, so I don't anticipate reviewing additional materials. I haven't requested it. To the extent I am provided additional materials, I certainly would consider them. If they were to change any of my opinions regarding the exposure or health-related opinions, I would submit separate addenda. But I don't anticipate that as I sit here.

MS. RAINES: Okay. Those are all the questions I have. Thank you for your time.

THE WITNESS: Thank you.

MR. PFAHL: Off the record.

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FURTHER EXAMINATION

BY MR. MILOTT:

Q Dr. Brodkin?

A Good afternoon.

Q Can you hear me okay, Doctor?

A Yes, thank you.

Q Doctor, once an individual is diagnosed with mesothelioma, subsequent exposures to asbestos are irrelevant to that disease progression; isn't that correct?

A I would agree with that. I mean, once a mesothelioma has developed, it really will depend on the biology of that tumor to a much greater extent than any ongoing exposure. I think that would be a de minimus effect once a mesothelioma has developed.

MR. MILOTT: Thanks, Doctor. Nothing further.

THE WITNESS: Thank you.

FURTHER EXAMINATION

BY MR. PFAHL:

Q Dr. Brodkin, just a few follow-up questions for you.

And in order to reorient ourselves, we had talked about the Brorby article, which was the joint compound reconstitution article. Do you recall that?

A Yes.

Q And then we reviewed an article that was a biopersistence study by Dr. Bernstein and also had one of the -- well, both of the articles had a Georgia-Pacific employee involved as well. Do you remember that?

A Yes.

Q With respect to the biopersistence paper that we discussed, you recall that they were looking at the biological response in the rats' lungs to exposure to pure chrysotile in one group and then joint compound dust that also included chrysotile in another group?

A Yes, the reconstructed joint compound.

Q Joint compound dust.

And they had found at the two termination points, the two study points, that there was an order of magnitude fewer chrysotile fibers in the combined exposure group as compared to the pure chrysotile exposure group.

Do you recall that?

A Yes, we discussed that.

Q Yeah, I'm just trying to reorient ourselves so that my follow-up questions make some sense.

A No, I recall that.

Q All right. And you recall that an observation they had was that there was an increased macrophage response in

the combined exposure group as opposed to the pure chrysotile group?

A Yes.

Q And that they found an increase in acidic environment due to that increased macrophage response in the combined exposure group as opposed to the pure chrysotile exposure group.

Do you recall that?

A That was discussed in the paper, yes.

Q All right. Would you expect there to be the same type of biological response in the human lung where you have human lung exposed to the combined exposure versus just pure chrysotile?

A Boy, that's a difficult question. I mean, even though we reviewed this in the findings, I haven't had a chance to read the article comprehensively. I would be very reluctant based on one toxicologic study to make a generalization that that's what happens in humans just because in one rat experiment they did observe that.

I think you would really have to observe that much more comprehensively probably in different rodents, different species, before you made some global conclusion about that.

I mean, obviously mechanistically one discusses that, but I don't think one knows that based on one

study.

And then, of course, the biologic implication one doesn't know either. Just because chrysotile is metabolized or kinetically reduced in number, it seems to be mechanistically as an increase in inflammatory response that can also cause injury, so what's the significance of that, and that we don't know in humans either.

Q Is there any reason why you think the human lung would react differently in terms of the macrophage response where you have joint compound dust and chrysotile being introduced into the lung which would be a greater particle burden than if you just had pure chrysotile fibers?

A Well, I guess the question is, you know, is this observation something that can be carried out into something that can be shown to be a definitive biologic response generally?

And the -- you know, I think this study is, you know, a hypothesis generating study. I mean, they've made this observation. From that, you would go to different species and try to replicate it and then perhaps consider some type of pathologic study where you looked at joint compound versus other exposures and see if there was a difference, although, it would be a

difficult study to design in humans.

But I think it's premature after one study to say this is what happens to humans. I just don't think you would do that on the basis of one animal experiment.

Q While it's just one study, does it raise in your mind an interesting question or hypothesis as to how the lung does react to a sanded product versus just a pure chrysotile exposure?

A I think it is an interesting finding, and that's why I say it's hypothesis generating. I mean, it makes you want to sort of do other studies and see if that's a true phenomenon. Maybe think of some different controls that would really address some of the other issues about whether this is truly chrysotile clearance or some other phenomenon on the basis of the particulates.

And then to do a study to see if that clearance is at the cost of increased inflammation that would have additional injury. Those are all things that could be done in follow-up.

So yes, I mean, I think they are interesting questions raised that are hypothesis generating.

MR. PFAHL: Thank you, Doctor. I appreciate your time.

THE WITNESS: Thank you.

(Signature waived.)

(Deposition concluded at 5:29 p.m.)

STATE OF WASHINGTON) I, Barbara Castrow, CCR, RPR,
) ss CCR #2395, a certified court
County of King) reporter in the State of
Washington, do hereby certify:

That the foregoing deposition of CARL A. BRODKIN, MD MPH FACOEM, was taken before me and completed on December 17, 2012, and thereafter was transcribed under my direction; that the deposition is a full, true and complete transcript of the testimony of said witness, including all questions, answers, objections, motions and exceptions;

That the witness, before examination, was by me duly sworn to testify the truth, the whole truth, and nothing but the truth, and that the witness waived the right of signature;

That I am not a relative, employee, attorney or counsel of any party to this action or relative or employee of any such attorney or counsel and that I am not financially interested in the said action or the outcome thereof;

That I am herewith securely sealing the said deposition and promptly delivering the same to Attorney Scott B. Pfahl.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my official seal this 21st day of December, 2012.

Barbara Castrow, CCR, RPR
Certified Court Reporter No. 2395
(Certification expires 11/24/12.)

EXHIBIT 2

2013 WL 214378
Only the Westlaw citation is currently available.
United States District Court,
D. Utah,
Central Division.

Linda SMITH, as Personal Representative
on behalf of the Legal Heirs of
Ronnie Smith, Deceased, Plaintiff,
v.
FORD MOTOR COMPANY, et al., Defendants.

No. 2:08–cv–630. | Jan. 18, 2013.

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Opinion

MEMORANDUM DECISION AND ORDER

DEE BENSON, District Judge.

***1** This matter is before the court on defendant Ford Motor Company's Daubert Motion to Exclude Expert Testimony of Samuel Hammar, M.D. (Dkt. No. 96.) The court heard oral argument on the motion on November 13, 2012. Having considered the parties' arguments, memoranda, and the relevant law, the court enters the following Memorandum Decision and Order.

BACKGROUND

Ronnie and Linda Smith, husband and wife, filed this asbestos personal injury action on July 15, 2008, in the Third District Court for the State of Utah. Ronnie Smith passed away on November 3, 2009, and the case is now being prosecuted by Linda Smith as plaintiff's representative on behalf of Ronnie Smith's heirs. The case was removed to this court on August 20, 2008.

In this action, plaintiff Linda Smith contends that Ronnie Smith was injured as a result of exposure to asbestos products which contributed to his development of mesothelioma. Plaintiff is suing numerous parties, including Ford Motor Company, which allegedly manufactured asbestos-containing products which exposed plaintiff to unknown doses of asbestos for unknown durations.

In his deposition, plaintiff Ronnie Smith stated he was exposed to asbestos-containing Ford brake parts while working as a part-time service station attendant at a full-service gas station in Cedar City, Utah, from August 1966 to May of 1968, a period of approximately 19 months. Although plaintiff offered that "he didn't know for sure" with respect to the number of times he changed brake pads on Ford vehicles, plaintiff asserts that during his 19 months of employment as a service station attendant, he may have changed brake pads on Ford vehicles on as many as seven occasions—and on one non-work related occasion in 1963 when he changed brake pads on his personal Ford automobile. According to Mr. Smith, the process of changing brake pads consisted of lifting the vehicles with a hydraulic-hoist and removing the vehicle's wheels with an air wrench. Thereafter, plaintiff would blow an accumulation of a black or gray dust-like substance away from the brake pad location with an air hose. Plaintiff claims the air hose caused the dust to enter the air which he then inhaled. After plaintiff removed the old brake pads and cleared the dust from the work zone, he replaced the brake pads, thereby completing the process. Plaintiff asserts that on the occasions he performed this process on Ford brand vehicles, the brake pads he was changing contained asbestos; and consequently, the dust he inhaled through the process of changing the brake pads exposed him to asbestos and caused his injuries.

DISCUSSION

Dr. Hammar's opinion is based on a theory of causation that has variously been described as the "every exposure" or "every breath" theory, which holds that each and every exposure to asbestos by a human being who is later afflicted

with mesothelioma, contributed to the formation of the disease.

Defendant asserts that this theory is without scientific foundation, that it is mere speculation designed for litigation, and that it is inadmissible pursuant to Rule 702 of the Federal Rules of Evidence and the standard set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.,* 509 U.S. 579 (1993).

***2** Having now reviewed the parties' briefing and the extensive number of exhibits, which includes prior court opinions, law review articles, and the declarations of numerous science and medical experts, including Dr. Hammar, the court agrees with the defendant's position. Dr. Hammar's opinion is, as a matter of law, unsupported by sufficient or reliable scientific research, data, investigations or studies, and is inadmissible under Rule 702.

Furthermore, even if it were deemed admissible under Rule 702, the court would exclude Dr. Hammar's testimony pursuant to Rule 403 of the Federal Rules of Evidence, because the probative value of such unsupported speculation by Dr. Hammar is substantially outweighed by the danger of unfair prejudice, as well as being confusing, and presenting a danger of misleading the jury. In reaching this conclusion, the court agrees with the growing number of published opinions from other courts that have reached a similar result: that the every exposure theory as offered as a basis for legal liability is inadmissible speculation that is devoid of responsible scientific support. When carefully examined, it becomes clear that Dr. Hammar's proffered testimony is precisely the kind of testimony the Supreme Court in *General Electric Co. v. Joiner,* 118 S.Ct. 136 (1997), observed as being nothing more than the "ipse dixit of the expert." *Id.* at 519.

The arguments in support of the court's holding are ably expressed in the opening and reply briefs of the defendant, and those briefs are adopted by the court, as well as is the expert opinion of Dr. Mark Roberts, which the court finds to be a well-reasoned examination of the standard *Daubert* factors most closely associated with the relevant medical scientific community. (See Def.'s Mem. in Supp. of Daubert Mot. to Exclude Expert Test. of Samuel Hammar, M.D., Def's Reply Mem. in Supp. of Daubert Mot. to Exclude Expert Test. of Samuel Hammar, M.D., Def.'s Mem. in Supp. of Daubert Mot. to Exclude Expert Test. of Samuel Hammar, M.D., Ex. 2, Mark A. Roberts M.D. Ph.D., Aff.) Consistent with those arguments, the court's reasons for rejecting Dr. Hammar's opinion include the following:

1. The every exposure theory is not based on sufficient facts or data.

The every exposure theory does not hold up under careful examination. It is questionable whether it can even properly be called a theory, inasmuch as a theory is commonly described as a coherent collection of general propositions used to describe a conclusion, and while there are some general propositions used by Dr. Hammar, they fall far short of supporting the legal liability he attempts to reach with them. Rule 702 and *Daubert* recognize above all else that to be useful to a jury an expert's opinion must be based on sufficient facts and data. The every exposure theory is based on the opposite: a lack of facts and data. When Dr. Hammar states that he cannot rule out any asbestos exposure as a possible cause of an individual's mesothelioma he is confirming the fact that there are insufficient facts and data to establish what minimum dosage levels of asbestos are required to cause cancer in a human being. The fact is the medical community at present does not know the answer to the all-important question regarding legal causation, how much is too much?

***3** Dr. Hammar seeks to base his causation opinion not on the thin reed that he cannot rule any exposure out, but on the opposite: he rules all exposures "in," boldly stating that Mr. Smith's mesothelioma "was caused by his total and cumulative exposure to asbestos, *with all* exposures and all products playing a contributing role." (Hammar Decl. at 17, ¶ 17.) This asks too much from too little evidence as far as the law is concerned. It seeks to avoid not only the rules of evidence but more importantly the burden of proof. It is somewhat like a homicide detective who discovers a murdered man from a large family. Based on his and other detectives' training and experience the detective knows that family members are often the killer in such cases. When asked if there are any suspects the detective says he cannot rule out any of the murdered man's relatives. This would be reasonable, but it would not allow the detective to attribute legal liability to every family member on the basis of such a theory. That is, in effect, what Dr. Hammar and plaintiff are trying to do here, without *any* underlying data as to the amount of chrysotile asbestos fibers found in Ford brakes that are needed to cause cancer in a human being.

Dr. Hammar wants to be allowed to tell a jury that all of the plaintiff's *possible* exposures to asbestos during his entire life were contributing causes of the plaintiff's cancer, and, therefore, sufficient to support a finding of legal liability

as to the manufacturer of each asbestos containing product, without regard to dosage or how long ago the exposure occurred. Just because we cannot rule anything out does not mean we can rule everything in.

2. Legal liability must rest on proof of specific causation.
Dr. Hammar's every exposure theory is supported by certain general conclusions that are not in dispute. These are: (1) asbestos fibers, both amphibole and chrysotile, are carcinogenic, (2) the majority (+/–80%) of mesothelioma cancers are caused by asbestos exposure, (3) prolonged and large exposures to asbestos in certain types of occupations and industries (e.g., mines, shipyards) satisfy the legal requirement of "substantial contribution" to allow a jury to determine legal causation in such large-dosage cases, and (4) there is no known minimum dose of asbestos that is required to cause cancer in a human being. Indeed, the vast majority of Dr. Hammar's declaration dwells on these matters about which the parties have no dispute.

What is missing from Dr. Hammar's proposed testimony is the necessary research and data to show that Mr. Smith's six alleged exposures to asbestos based on six brake jobs in the 1960s constitutes proof of sufficient exposure to cause Mr. Smith's cancer on its own (which plaintiff has never contended) or that such exposure amounted to more than an insignificant or de minimus factor in the development of the disease that afflicted Mr. Smith forty years after those exposures.

***4** Dr. Hammar cites to no studies, reports, examinations, or data of any kind to show that the alleged dust that Mr. Smith allegedly breathed in during those six brake jobs was sufficient to be a contributing cause, substantial or otherwise, to the development of Mr. Smith's cancer. He points to no research or findings that even suggest that the amount of chrysotile fibers that one would expect to find in the alleged brake dust associated with those incidents would cause cancer, let alone how many of such fibers would still be carcinogenic after the brakes had been used long enough to need to be replaced. Dr. Hammar cites to no such studies because there are none.

Dr. Hammar's testimony does virtually nothing to help the trier of fact decide the allimportant question of specific causation. His opinions are based solely on his belief that he should not rule out any exposure as a contributing cause. Accordingly, with regard to the most basic purpose of Rule 702—that the expert's opinion should be helpful to the jury—Dr. Hammar's testimony fails.

Beyond the almost complete lack of facts or data to support specific causation, Dr. Hammar's testimony also appears on its face to be inconsistent. He tells us on page 4 of his declaration that "I do not believe every asbestos fiber an individual breathes into their lungs contributes to the development of mesothelioma," and on page 5 that "it is not possible to specifically identify an individual fiber from the individual's occupational, non-occupational, or bystander exposure that caused the cellular events that led to the development of mesothelioma," yet concludes his opinion on page 17 with the bold declaration that "[i]n my opinion, the information referenced in my report is sufficient for me, as a practicing pathologist to come to the specific causation determination to a reasonable degree of medical certainty that each of defendant's product [sic] was a contributing cause in the development of Mr. Smith's mesothelioma and death." Hammar Decl. at 4–5, 17

3. Dr. Hammar's and Plaintiff's references and citations are, for the most part, irrelevant.
In his declaration, Dr. Hammar cites to numerous scholarly studies and articles in support of his testimony. Upon even a cursory review, virtually all of these sources are either irrelevant or of little assistance to the court in providing support for the admissibility of Dr. Hammar's opinions. For the most part they only reinforce the general undisputed principles discussed above.

The same is true for the majority of plaintiff's memorandum in support of Dr. Hammar's testimony. All of the separate bullet-points from pages 6 to 14 relate to large scale exposures of asbestos in mills and plants and other work sites that have nothing in common with this case.

4. The scientific literature and studies that exist do not support Dr. Hammar's views.
Separate and apart from the lack of any reliable scientific methodology or data that supports Dr. Hammar's opinion, the research that has been done contradicts his point of view. Chief among these are the tests that have been conducted to determine if there is any greater incidence of mesothelioma cancers among auto mechanics than in the general population. All of these studies have shown no statistically significant difference.

***5** Plaintiff discounts these studies as worthless because they were funded by asbestos manufacturers, but has not shown them to be flawed or fraudulent. And perhaps more to the point, neither plaintiff nor his expert reference any studies to the contrary.

5. Numerous courts have examined the every exposure theory and found it lacking under *Daubert* and Rule 702. Numerous courts have examined and rejected expert testimony attempting to assert causation without assessing the dose and held the every "exposure theory" lacking under Daubert and Rule 702. These include:

Moeller v. Garlock Sealing Technologies, LLC, 660 F.3d 950, 952 (6th Cir.2011)

Lindstrom v. A–C Prod. Liab. Trust, 424 F.3d 488 (6th Cir.2005)

Wills v. Amerada Hess Corp., 379 F.3d 32, 40, 53 (2d Cir.2004)

Borg–Warner Corp. v. Flores, 232 S.W.3d 765, 774 (Tex.2007)

Georgia–Pac. Corp. v. Stephens, 239 S.W.3d 304, 321 (Tex.App .2007)

Smith v. Kelly–Moore Paint Co., Inc., 307 S.W.3d 829, 839 (Tex.App.2010)

Butler v. Union Carbide Corp., 310 Ga.App. 21, 712 S.E.2d 537 (2011)

Betz v. Pneumo Abex, LLC, 44 A.3d 27, 58 (Pa.2012)

In re Toxic Substances Cases, A.D. 03–319, 2006 WL 2404008 (Pa.Com.Pl. Aug. 17, 2006)

This court agrees with the general assessment of these various state and federal courts that the every exposure theory does not qualify as admissible expert testimony. The *Butler* court summed up expert testimony regarding the every exposure theory accurately by stating that an expert's "any exposure theory is, at most, scientifically-grounded speculation: an untested and potentially untestable hypothesis." *Butler v. Union Carbide Corp.,* 310 Ga.App. 21, 43, 712 S.E.2d 537, 552 (2011), cert. denied (Oct. 17, 2011)

CONCLUSION

For the foregoing reasons, Ford Motor Company's Daubert Motion to Exclude Expert Testimony of Samuel Hammar, M.D. is GRANTED.

End of Document

EXHIBIT 3

SUPERIOR COURT OF THE STATE OF WASHINGTON
KING COUNTY

FREE,

Plaintiff,

vs.

AMETEK et al,

Defendants.

CAUSE NO. 07-2-04091-9 SEA

RULING ON MOTION IN LIMINE UNDER *FRYE V. UNITED STATES*

Signed this 29th day of February, 2008.

Judge Suzanne M. Barnett

Exhibit F
to Union Carbide Corporation's Frye Motion

ORIGINAL

Free v. Ametek, et al., 07-2-04091-9 SEA
February 28, 2008

This is the court's ruling on motions *in limine* brought under the case of *Frye v. United States,* 293 F. 1013 (D.C. Cir. 1923). The issue before the court is the admissibility of testimony by certain of plaintiff's designated expert witnesses regarding the proximate cause/s of plaintiff's mesothelioma. Specifically, plaintiff's experts seek to testify that once a product is identified and exposure is established, any level of exposure greater than ambient levels or greater than 0.1 fibers per cubic centimeter of air per work-year is a substantial factor, undifferentiated and incapable of differentiation, in proximately causing plaintiff's disease.

In this case, defendant Caterpillar, Inc. ("Caterpillar") made two motions to limit or preclude testimony by plaintiff's designated experts. The first motion *in limine* seeks to preclude Dr. Carl Brodkin from testifying that every exposure to asbestos, above ambient levels, is a substantial factor in causing plaintiff's asbestos-related disease, which in Mr. Free's case is malignant mesothelioma. Caterpillar's second motion *in limine* seeks to preclude Dr. Samuel Hammar from testifying that plaintiff's asbestos exposure from any defendant's products was a substantial factor in the development of his mesothelioma unless plaintiff can establish that his exposure to that defendant's product/s was greater than 0.1 fibers per cubic centimeter year ("fbrs/cc yr")[1]. Caterpillar also moved *in limine* to exclude testimony of plaintiff's experts on other grounds. The court does not deal with those specific grounds in this ruling. This ruling is an analysis under the *Frye* case only.

Defendant Cameron International Corporation[2] moved *in limine* for exclusion of testimony by Dr. Brodkin relating to marine engineering, specifically the design and function of Cameron's GND-8 engines; whether the GND-8 engines were designed for use with other systems, including exhaust systems; what Cameron knew or should have known about the installation of its engines in a particular context, specifically the methods of exhaust or the nature of exhaust systems that would or would not be attached to the engines; the varieties and methods of insulation used by the U.S. Coast Guard in 1959-60; the state of the art of burn protection measures for crew members; and whether a warning on the Cameron engine would have been useful. Cameron's attempt to limit Dr. Brodkin's testimony on these issues implicates a *Frye* analysis only to the extent Dr. Brodkin asserts that these areas are within the specialized knowledge, skill, training, and experience of an occupational medicine physician.

The court heard two and a half days of testimony and argument on the *Frye* motions. Plaintiff's challenged experts, Drs. Hammar and Brodkin, testified, as did defendants' experts, Dr. David Garabrandt, Captain Richard Silloway, USN (ret.), and Dr. Suresh

[1] "fbrs/cc yr" refers to the number of fibers present in one cubic centimeter of air over a period of 8 hours per work day of exposure, for 250 work days per year.
[2] Cameron International Corporation is the successor in interest to Cooper-Bessemer, the manufacturer of the GND-8 engine at issue. For simplicity, the defendant will be referred to as "Cameron".

Moolgavkar. Caterpillar called Dr. Garabrandt, an epidemiologist who is board certified in, *inter alia*, occupational medicine, and Dr. Moolgavkar who is qualified as, *inter alia*, a biostatistician. Captain Silloway, called by Cameron, is a marine engineering expert.

Frye standard

The *Frye* court probed the interstice between the "experimental and [the] demonstrable stages" of science as it relates to the admissibility of expert testimony in a court of law. The rule of *Frye*, correctly stated by counsel in this case, is that an expert's testimony on or deductions (notably, not "inductions") from science in this "twilight zone" must be based upon a principle or discovery that is "sufficiently established to have gained general acceptance in the particular field in which it belongs."[3]

Those few but dense words from the *Frye* court have spawned a generation (or two) of jurisprudence on the issue of expert scientific testimony.

Rule 702 of the Washington Rules of Evidence establishes that expert opinion testimony is admissible if an expert, qualified by "knowledge, skill, experience, training, or education" can offer "scientific, technical, or other specialized knowledge" that will "assist the trier of fact to understand the evidence or to determine a fact in issue". Notwithstanding a witness's abilities, however, Washington courts have adhered to the *Frye* test for admissibility of opinions in both civil and criminal cases.

Dr. Samuel Hammar

Dr. Hammar is a preeminent pathologist concentrating in pulmonary pathology. He has extensive relevant training in his field and many years of clinical experience with patients presenting with asbestos-related diseases. Dr. Hammar testified that he has seen over 6,000 patients with asbestos-related diseases. He serves on an international mesothelioma review panel. He has a special interest in, and has studied, the pathology of cancers in particular. Dr. Hammar is qualified by both training and experience to testify as an expert in pulmonary pathology and asbestos-related disease.

Dr. Hammar testified, and defendants did not challenge, that mesothelioma is a "dose-response" disease, that is, that there is a positive correlation between increased concentration of exposure and risk of development of the disease. The parties also do not dispute that mesothelioma is a disease with a protracted latency, in a wide range of ten to forty years.[4] Dr. Hammar further testified that it is cumulative exposure, over the work life of the patient, that accounts for the development of mesothelioma. Once a patient develops mesothelioma it is impossible to determine which specific exposure, either by dose, type, or time, caused the disease.

Dr. Hammar did not purport to have any expertise in determining intensity of exposure in any particular setting. He did concede that some exposures are less intense than

[3] 293 F. at 1014.
[4] The actual range is not relevant to the issues in this case or in the *Frye* motions.

others. He further testified that chrysotile asbestos fibers have a very short half life (perhaps 90 – 180 days) and clear the lungs over time; whereas other types of asbestos fibers have a much longer, by orders of magnitude, half-life. Nonetheless, inasmuch as asbestos molecules carry no labels or tags, by the time a pathologist is studying tumor cells, there is no way to know what type of fiber or what amount of fiber initiated the cellular changes that lead to mesothelioma.

The aspects of Dr. Hammar's testimony challenged by defendants in this case are his conclusions that *1)* because mesothelioma is a dose-response disease, and because of its latency, it is undifferentiated cumulative exposures that cause the disease; and *2)* every exposure to asbestos can and should be considered a substantial factor contributing to the development of mesothelioma.

During his testimony, Dr. Hammar conceded that an exposure would have to be at a level of at least 0.1 fbrs/cc yr to be considered a contributing factor. Dr. Hammar also conceded that his opinion is a hypothesis, not a scientific conclusion. As support for his opinion, Dr. Hammar relied on various studies and regulatory analyses. The regulatory standards are not probative of scientific analysis or acceptance in the scientific community. The epidemiological studies and meta-analyses do not analyze cases of exposures at very low levels.

The assumption of some epidemiologists and practitioners in the field of asbestos-related diseases is that the risk of occurrence at low levels of exposure follows a straight line below the level of available data. This downward extrapolation of a straight line correlation between exposure and risk of development of mesothelioma is, however, not proved by empirical data. In fact, according to defendants' biostatistician, Dr. Garabrandt, just the opposite is true. Referring to the meta-analysis performed by Hodgson and Darnton,[5] he and they conclude that a straight-line correlation is not accurate for the data that are available, let alone for extrapolation to data that are not collected.[6]

Conventional wisdom is that there is no safe level of exposure to asbestos. A more accurate statement of conventional wisdom, however, would be that there is no *known* safe level of exposure, just as there is no known threshold level for causation of asbestos-related disease. Dr. Hammar's hypothesis, therefore, while persuasive in lay, "common sense" terms, is not supported by replicable, scientific methodology. While it may be *assumed* to be accurate and sufficient for purposes of connecting asbestos exposure to mesothelioma in general, the assumption that every exposure to asbestos over a life's work history, even every exposure greater than 0.1 fbrs/cc yr, is a

[5] J. Hodgson, A. Darnton, "The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure," 2000. At page 577, the authors note that the data are graphed as linear, but on a log scale. An actual plotting of the data points would result in a curve, not an applied straight-line.
[6] "Direct statistical confirmation of a threshold from human data is virtually impossible." "[W]e do not believe there is a good case for assuming any threshold for mesothelioma risk." Hodgson & Darnton at p.583. No threshold for risk precludes a conclusion that the same data predict a threshold for causation.

substantial factor contributing to development of an asbestos-related disease, is not a scientifically proved proposition that is generally accepted in the field of epidemiology, pulmonary pathology, or any other field relevant to this case.

There is no *known* threshold; there is no *known* safe level of exposure. That does not mean none exists; it simply means modern science has not and cannot, with current scientific expertise or relying on existing studies, determine what that level of exposure is. Dr. Hammar may not testify that any exposure at the level of 0.1 fbrs/cc yr or ~~greater~~ *less* is a substantial contributing factor to the development of mesothelioma.

Dr. Carl Brodkin

Dr. Brodkin is an occupational medicine physician; that is, a physician who specializes in the assessment of exposure-related diseases. His *curriculum vitae* reveal extensive knowledge, training, and experience in the field of occupational and environmental medicine. He is qualified to testify as an expert in occupational medicine.

Dr. Brodkin testified that it is generally accepted practice in the field of occupational medicine to take a careful and detailed occupational history. The conducting of the history is emphasized in training occupational and environmental medicine physicians and in their practice. He also testified that, based upon such an occupational history and certain studies evaluating the level of exposure in selected occupations, an occupational medicine physician can and does estimate the level of exposure of any particular patient. The exquisite occupational history prepared by an occupational medicine practitioner may be a generally accepted scientific practice in that field, but an occupational history of a patient cannot establish causation with regard to exposure to any specific fiber type from any specific fiber source.

At the *Frye* hearing, Dr. Brodkin repeated the opinion, now accepted in medicine and borne out by epidemiological studies of risk, that mesothelioma appears to be a dose-response disease. The epidemiological studies deal with statistical risks based on estimates of cumulative exposure over the life of the patient. As segregation of specific causes is not currently scientifically possible, physicians look upon mesothelioma as resulting from cumulative exposure.

Dr. Brodkin opined that, therefore, every biologically significant exposure to asbestos, that is, every exposure above ambient levels[7] within the latency period for mesothelioma is a proximate cause of the disease. Because medical science has only been able to determine that mesothelioma is related to cumulative exposure, Dr. Brodkin concludes that each component of that cumulative exposure is a necessary and substantial factor in development of the disease.

[7] Dr. Brodkin defined the public's "fresh" air as "background." He defined "ambient" levels as those that might be present in a workplace, whether or not the patient is working directly with asbestos products.

Scientific studies do clearly show that risk of developing the disease increases with increased exposure. Determination of risk in an epidemiological study is not, however, an assessment of causation in a particular case. Downward extrapolation from the studies that establish levels of risk at doses at or above 10 fbrs/cc yr is not a sound scientific methodology and is not generally accepted in the field of epidemiology or occupational medicine.

To support his opinion that every biologically significant exposure is a substantial factor contributing to or causing development of disease, Dr. Brodkin analogized a series of exposures to the chapters of a book – each chapter contributes to the whole. Alternatively, he used the bucket analogy. He opined that it doesn't matter if you add water to a bucket by the teaspoonful or by the gallon, the water in the bucket is all the same and when the bucket is full it doesn't matter how it got that way. He makes no allowance for insignificant chapters in a book, pages inserted in error by the publisher, or excess materials between the covers, such as flysheets or title pages. Further, with his bucket, he makes no allowance for the temporal effects of evaporation or condensation or the possibility of contamination or spillage.

Dr. Brodkin's analogies are not good science and they do not make good law.

Dr. Brodkin will not be permitted to testify that every biologically significant exposure to asbestos above ambient levels is an undifferentiated proximate cause of mesothelioma. We do not know, and modern science cannot tell us what a biologically significant exposure is. We cannot tell which fiber or group of fibers from which sources at what time in the life of a patient overwhelmed that patient's individual bodily defenses.

Dr. Brody

Dr. Brody did not testify at the *Frye* hearing, but his deposition in this case was tendered to the record. To the extent Dr. Brody would opine that "every fiber" or every undifferentiated exposure to asbestos, regardless of type, level, or time of exposure is a substantial factor in causing mesothelioma, that testimony is excluded.

Conclusion

In arguing the *Frye* motions in this case, plaintiff suggested that a *Frye* analysis was inapposite. Citing *Lockwood*[8], *Mavroudis*[9], and *Hue*[10], plaintiff argued that the science offered by his experts is not novel, but is rather accepted and part of the legal record in this state. In fact, none of the cases upon which plaintiff relies is the result of a *Frye* inquiry. Further, none of those cases reaches the point central to this case.

[8] *Lockwood v. AC & S, Inc.*, et al., 109 Wash.2d 235 (1987).
[9] *Mavroudis v. Pittsburgh-Corning Corp.*, et al., 86 Wash. App. 22 (Div. I, 1997).
[10] *Hue v. Farmboy Spray Co., Inc.*, et al., 127 Wash.2d 67 (1995).

Lockwood, a 1987 Supreme Court case, establishes the threshold for product identification and factors to consider in determining whether exposure is sufficient to tender the causation question to a jury. In 1997, in *Mavroudis,* Division I of the Court of Appeals altered the standard for determination of proximate cause of asbestos-related disease. In light of the apparent statistical relationship between cumulative exposure and risk of development of disease, the *Mavroudis* court determined that the "substantial factor" test should replace the "but-for" test.[11]

Plaintiffs also argued the *Hue* case, analogizing the "cloud of poison" dispersed in that case to cumulative exposures to asbestos. *Hue* is a cumulative exposure case against wheat farmers whose chemical crop dust caused harm to the downwind neighbors' plants. The *Hue* court found that it was impossible to segregate which particular chemicals in the cocktail caused the damage, as each contributed to the cloud of poison set loose by the crop duster.

Mavroudis acknowledged the *Hue* authority. The Court of Appeals specifically stated that

> while the substantial factor test may be unclear with regard to an *insubstantial* cause that combines with other causes to produce an injury, we need not reach that issue in this case.

86 Wash. App. at 30-31 *(emphasis in original)*. The court noted that the Supreme Court, after *Hue*, might require a different instruction than the one approved in *Mavroudis,* but the Supreme Court has not spoken on the issue. It is that mystery, therefore, of what is substantial and what is insubstantial that raises the *Frye* issue in this case[12].

Plaintiff's reliance on these reported cases is insufficient. To overcome the unknown in this asbestos case, an analogy to crop dusting is not persuasive. In *Hue* the parties were unable to determine which chemical caused the damage. There was a series of chemical applications, but it was not the repetition that rendered causation undifferentiated, it was the chemical composition of each offending cloud.

In asbestos, a plaintiff is not exposed to all defendants' products simultaneously and *seriatim*. Each exposure varies by type, level, and extent. Science cannot trace the pathology of the asbestos-related disease back to "fiber X". Nor can it say at what point in the latency of the disease that fiber or group of fibers triggered cellular change in an

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[11] *Mavroudis* addressed specifically the propriety of a substantial factor jury instruction. The instruction at issue introduced the substantial factor principle. It is important not to lose sight of the instruction at issue. The challenged jury instruction required the jury to determine that any individual cause "operating alone would have been sufficient." 86 Wash. App. at 30, n.5.

[12] *Mavroudis* cites the analogy made in W. Keeton, D. Dobbs, R. Keeton, & D. Owen, *Prosser & Keeton on Torts*, § 41 (5th ed. 1984) regarding the substantial factor test of proximate cause. The substantial factor test is designed to exclude liability for insubstantial factors *("throwing a match into a forest fire")*. 86 Wash.App. at 32.

individual. We do not know, for example, whether, after a hiatus or a period of low-dose exposure to a fiber with a shorter half-life, the body might heal itself, only to succumb at the next exposure.

We do not know, and science cannot tell us.

Cameron

Cameron filed a motion to exclude testimony by Dr. Brodkin relating to marine engineering issues. Dr. Brodkin testified that, as part of a thorough occupational history, it is generally accepted practice in the field of occupational medicine to inquire as to warnings posted for, or safety precautions taken by, the patient. To the extent Dr. Brodkin seeks to testify to this inquiry, such testimony is within his field of expertise.

The remainder of Cameron's motion is more properly characterized as a motion to exclude under ER 702. Having heard the testimony and argument, however, the court will make the following ruling.

Dr. Brodkin will not be permitted to testify as to engine design, construction, or employment. He will not be permitted to testify as to exhaust temperature levels, exhaust systems that were or were not or might have been or might not have been attached to the Cameron engines at issue in this case. He will not be permitted to testify as to what Cameron knew or should have known. He may not testify as to whether, in what way, or with what recommendations or precautions, Cameron should have affixed any warnings to its engines.

EXHIBIT 4

2006 WL 2404008
Only the Westlaw citation is currently available.
Court of Common Pleas of
Pennsylvania, Allegheny County,
First Judicial District, Civil Trial Division.

In re: TOXIC SUBSTANCE CASES
Incorporated by reference: A. John Vogelsberger and Freda M. Vogelsberger, his wife Plaintiffs,
v.
Owens-Illinois, Inc., et al., Defendants.
Tammie L. Cline, Administratrix of the Estate of Michael Cline, Deceased, and Tammie L. Cline, in her own right, Plaintiff,
v.
Pneumo Abex LLC, et al., Defendants.
Charles Simikian, Plaintiff,
v.
Pneumo Abex LLC, et al., Defendants.
Clinton M. Bahnemann and Susan K. Bahnemann, his wife, Plaintiffs,
v.
Allied Signal, Inc., et al. Defendants.

No. A.D. 03-319. | No. GD 02-018135, 05-010028, 05-004662, 04-010451. | Aug. 17, 2006.

Opinion

OPINION FOLLOWING FRYE HEARING

COLVILLE, J.

PROCEDURAL HISTORY

***1** In the Spring of 2005, DaimlerChrysler and Volkswagen of America filed a Global Motion for Frye Hearing requesting that this court [1] entertain arguments challenging the general acceptance of the methodologies that defendants anticipated would be asserted in support of plaintiff's experts' causation opinions against friction product manufacturers in all pending cases. Several additional friction product manufacturer defendants joined in this Motion. The challenged methodologies were those that defendants asserted were regularly and historically employed by plaintiff's experts against friction product manufacturers in past asbestos cases. [2] These methodologies purportedly supported the opinions offered by plaintiff's experts asserting that exposure to friction products was a medical cause of asbestos-related disease in specific plaintiffs.

Because I was not satisfied that I could properly conduct a Frye challenge and analysis as to "all pending cases," I directed the lawyers from the three local law firms representing plaintiffs in asbestos cases and all lawyers representing any friction product manufacturer defendant in asbestos cases in Allegheny County, to designate "a handful of representative cases" within which specific Frye challenges might be properly raised, and resolved. [3] Once those cases were identified, this court directed plaintiffs' counsel to file expert reports related only to medical causation theories that would be relied upon by each plaintiff at trial. The expert reports were expected to identify the opinions, and the basis for the opinions anticipated to be offered by plaintiff's experts at trial, including, in particular, the opinions and methodology supporting the plaintiff's theory that exposure to friction products was a proximate cause of the plaintiff's asbestos-related disease.

In response to this direction, plaintiffs filed non-case-specific expert reports [4] offered by Dr. Maddox and Dr. Laman. In their expert reports, both Dr. Maddox and Dr. Laman offer the opinion that each plaintiff's exposure to each of the defendant's friction products was a proximate cause in the development of the plaintiff's asbestos-related disease. Ultimately, these opinions are grounded upon the proposition asserted by Drs. Maddox and Laman, that every single exposure to every asbestos product is a proximate cause of a subsequently diagnosed asbestos-related disease. Reliance upon this proposition ultimately, and necessarily, supports Dr. Maddox and Dr. Laman's opinions, (offered to a reasonable degree of medical certainty), that each of the plaintiff's exposures to each friction product was a substantial contributing factor, i.e. proximate cause, of the plaintiff's subsequently diagnosed asbestos-related disease.

THE FRYE CHALLENGE

Defendants challenged the general acceptance of any methodology that would support the medical causation opinions offered by Drs. Maddox and Laman in numerous respects. In the judgment of this court, however, the only

question that need be resolved for purposes of this appeal is whether Drs. Maddox and Laman's ultimate opinion-that every exposure constitutes a proximate cause of a subsequently diagnosed asbestos-related disease-is based upon generally accepted methodologies in the relevant scientific field. In my opinion, based upon the evidence of record, it is not.

NOVELTY

***2** On August 17, 2005, this court entertained argument regarding the novelty of the opinions expressed by Drs. Maddox and Laman in their expert reports. Following that hearing, I concluded that the opinions offered by Drs. Maddox and Laman were, in fact, novel and that a Frye hearing was warranted.

THE HEARING

I conducted a Frye Hearing in the four identified cases on October 17, 18, and 21 of 2005. Subsequent testimony was concluded, outside of my presence, but submitted by transcript for review, by the end of 2005. In addition, the parties agreed to submit the prior testimony of numerous witnesses from other court proceedings and many scientific papers, industrial/commercial/trade documents, governmental publications, and other papers, documents, and publications referenced and relied upon by the witnesses in support of their respective positions. In short, the testimony and scientific literature, submitted to and reviewed by this court, is voluminous.

It is appropriate to note that the legal, medical, and scientific issues raised and implicated by this *Frye* challenge have been exhaustively and capably briefed by the litigants, and are a part of this record. Any effort by me to provide greater clarity to the status of the law, medicine, or scientific consensus or disagreement on the issues involved would be in vain. As such, while this opinion is offered as a modest attempt to assist the appellate court in a meaningful review of the proceedings before this court, and also to convey to the litigants the primary and fundamental considerations of this court in arriving at its conclusion, any attempt to exhaustively describe this court's considerations would be imprudent, if not impossible.

In resolving this *Frye* challenge I have considered the testimony of the witnesses, voluminous scientific literature, and numerous legal authorities proffered in support of the plaintiffs' and the defendants' respective positions. In the end, my decision ultimately rests upon whether the plaintiffs experts' opinions were based upon methodologies utilizing discrete and specific scientific principles logically applied in a manner that can be affirmatively articulated, referenced, reviewed, and tested, and empirically verified or whether the testimony was based upon the "best estimate," the "gut instinct," or the "educated guess" of the experts. Thorough review of the transcripts and the various authorities relied upon by the plaintiffs' experts persuades me that the plaintiffs' experts' foundational opinions are based upon the latter rather than the former. [5]

FRYE STANDARD APPLIED

Specifically, I precluded Drs. Maddox and Laman from testifying that each and every exposure to asbestos is a substantial contributing factor in the development of asbestos related disease and that the specific plaintiff's disease in this case was caused by exposure to a specific defendant's friction product. I did so because I discern no generally accepted methodology within the relevant scientific field to support those opinions. *Grady v. Frito-Lay, Inc. 839 A.2d 1038 (Pa.2003),Trach v. Fellin,* 817 A.2d 1102 (Pa.Super.2003).

THE FOCUS ON METHODOLOGY [6]

***3** It is important to recognize two fundamental terms as defined by the Superior Court. The first term, methodology, is "[1] a method of research in which a problem is identified, [2] relevant data are gathered, [3] a hypothesis is formulated from these data, and [4] the hypothesis is empirically tested." *Trach,* 817 A.2d at 1113. "Empirical" is defined as "provable or verifiable by experience or experiment." *Id.* (citations omitted). A vital characteristic of the scientific method, as the Superior Court determined, consists of the "ability to test or verify a scientific experiment by a parallel experiment or other standard of comparison (control) and to replicate the experiment to expose or reduce error."

> One of the primary reasons we embraced the Frye test ... was its assurance that judges would be guided by scientists

> when assessing the reliability of a scientific method. Given the ever-increasing complexity of scientific advances, this assurance is at least as compelling today as it was in 1977, when we decided that case. We believe now, as we did then, that requiring judges to pay deference to the conclusions of those who are in the best position to evaluate the merits of scientific theory and technique when ruling on the admissibility of scientific proof, as the Frye rule requires, is the better way of insuring that only reliable expert scientific evidence is admitted at trial.

Grady v. Frito-Lay, Inc. 839 A.2d 1038 (Pa.2003) (citations omitted)

THE UBIQUITY OF ASBESTOS [7]

Asbestos is everywhere. Everyone is exposed to asbestos. Everyone has asbestos in his or her lungs. Individuals without specific occupational exposure to asbestos can be expected to have hundreds of thousands of asbestos fibers in their lungs. Asbestos is in the air. It comes from a multitude of products which are incorporated everywhere into modern life. Asbestos occurs naturally in the ground and is naturally released from rock outcroppings. Humans would be exposed to asbestos even if it had never been incorporated into industrial products.

This exposure, to which every human being is subjected, is often, and alternatively, referred to as “background exposure” or “ambient exposure”. For instance, experts suggest that the average ambient exposure in Pittsburgh is approximately .0001 fibers per milliliter of air. Consistent with this exposure, one would expect to find, on average, one fiber of asbestos in every 10 liters of air on every street corner in Pittsburgh. No one, including the plaintiff's experts, proffers an opinion that this level of exposure creates an increased risk of the development of any asbestos-related disease. Accordingly, this background or ambient exposure is simply not sufficient to allow experts to causally attribute asbestos-related disease to it. Everyone, including the plaintiff's experts, agrees that something greater is required. The argument in this *Frye* challenge, in part, revolves around the question of how much greater quantity of exposure is necessary to permit the causal attribution of an asbestos-related disease to a particular asbestos exposure.

GENERAL AND SPECIFIC CAUSATION

***4** The ultimate question to be resolved is whether the opinions offered by Drs. Maddox and Laman are supported by generally accepted methodologies within the relevant scientific fields. The specific opinion, whose supporting methodology is being questioned, is that the asbestos related disease suffered by the specific plaintiff[s] in this case was proximately caused by exposure to a specific friction product manufactured by a specific defendant. This opinion ultimately rests upon the proposition and opinion that each and every exposure to the defendant's friction products constituted a proximate cause of the specific plaintiff's subsequently diagnosed asbestos-related disease.

In order to meet their burden plaintiffs must offer evidence, (presumably in the form of expert medical testimony), from which a jury can reasonably find or infer, that *the specific plaintiff involved in this case* was, *in fact,* proximately caused to develop an asbestos-related disease as a result of the plaintiff's inhalation of fibers shed from a specific friction product of the defendant.

Of course, there is no direct, (i.e observational), evidence of this, and no direct evidence can be plausibly expected. Such evidence is simply not practically available because of restrictions on human capacities for observation and knowledge. It is, as a practical fact, impossible to follow a single fiber shed from a specific defendant's product into the airway of a specific plaintiff, and watch it interact with the biological structures of the human body, and thereby cause a disease. Accordingly, the plaintiffs must rely upon expert medical opinion testimony to develop evidence from which a jury can reasonably infer that *the plaintiff in this case* was, *in fact,* caused to be injured by a specific defendant's product. [8]

Drs. Maddox and Laman begin by stating their opinion on the question of general causation-that all asbestos fibers can potentially cause disease. [9] This opinion, standing alone, is not sufficient evidence from which a jury can reasonably find or infer proof of specific causation, i.e. that the plaintiff in this case was, in fact, caused to develop asbestos-related

disease as a result of exposure to a specific defendant's friction product.

CASE REPORTS

Plaintiff's counsel has repeatedly assured this court that they do not proffer the opinions of Drs. Maddox and Laman as supported by case reports alone, but argue that other generally accepted methodologies support their experts' opinions. Notwithstanding this assurance, plaintiff's counsel has also repeatedly suggested that reliance upon case reports of brake/auto repairmen suffering from asbestos related disease is one "arrow in the quiver" of their expert's generally accepted methodology. In this respect a word or two regarding case reports is warranted.

Case reports are nothing more than reports by other physicians and professionals confirming the development of a disease in an individual patient with additional information about that patient. For instance: "John Doe, male, 6ft 7 in., 78*l*bs, smoker, coffee drinker, astronaut, and Socialist is diagnosed with lung cancer." constitutes, an admittedly sketchy, case report. Case reports can be valuable because as they grow in number, physicians can begin to develop hypotheses regarding the correlations and associations between the disease and other known factors. If many people who develop lung cancer are smokers, a hypothesis that smoking causes lung cancer may be generated. But the development of this hypothesis alone is not a generally accepted methodology that would support the opinion that smoking actually causes cancer-or, more importantly, that smoking caused a specific plaintiff's cancer, until the hypothesis is tested and validated through the scientific method that requires repeatable, testable verification. Utilizing an unverified hypothesis to support a causal attribution opinion is not generally accepted methodology.

***5** The reason case reports (even multiple case reports) cannot, alone, support a causal attribution opinion is because they only report associations-not causal correlations. Sometimes an association exists because there is a causal correlation. Sometimes associations exist because there is a coincidence, and nothing more. If, for instance, we learned that several case reports, like that of our "John Doe" above, were being reported we might conjecture that any of a number of habits, conditions, and beliefs: being male, very thin, a smoker, a coffee drinker, an astronaut, and a Socialist cause lung cancer-and while our conjecture would be supported by several associations-we would be wrong in most instances.

Of course, other diagnostic criteria might assist us-for instance, we could rule out "being a Socialist" as a cause because of an absence of a biologically viable mechanism to support the hypothesis that "being a Socialist" causes lung cancer. Of course, we would continue to conjecture that the other attributes were causal, and we would be wrong. Additional study may identify anomalies in the statistical data, such as inadequate representation of a specific group, requiring the removal of another attribute-say "being an astronaut". Additional case reports balancing out early aberrational results may demonstrate that "being male" and "being very thin" really are not associated after all.

In the end, after review of countless case reports we would be left with the situation, as it appears to actually be today-there are significant associations between coffee drinking and lung cancer, and between smoking and lung cancer. If we relied solely upon the case reports we would quite likely conjecture that both smoking and coffee drinking cause lung cancer-and we would be wrong. We would be wrong because only one is a causal correlation-smoking causes lung cancer. In spite of all the case reports that suggest a connection between coffee drinking and lung cancer, the connection is only a coincidental association-coffee drinking does not cause lung cancer. [10] We would have been wrong not because science failed us, but because we failed science. We would have been wrong because we failed to utilize the scientific method to distinguish a coincidental association from a causal correlation. Importantly, for our purposes in this *Frye* challenge, our failure to use the scientific method renders our errant causal attribution opinion inconsistent with generally accepted methodology within the relevant scientific field. For this reason, our errant opinion, and underlying methodology, would not have (and should not have) survived a *Frye* challenge.

The scientific method's requirement of empirical verification saves us from the peril of confusing "coincidental association" with "causal correlation". Case reports alone, or in conjunction with other methodology short of empirical verification, do not meaningfully support the plaintiff's expert's opinions.

DOSE RESPONSE CURVE

***6** Next, Drs. Maddox and Laman state their reliance upon the generally accepted consensus, (if not fact), that all asbestos-related diseases are, at least in some respects, (i.e. at high levels of exposure, or "high dose" exposures [11]) subject to a dose-response curve. That is to say that (at high dose exposures), greater amounts of asbestos fibers inhaled into the lungs or other biological structures and retained there, in some manner, correlates to a greater probability of developing an asbestos-related disease. Given the applicability of a dose response curve, one can reasonably assume, all other things being equal, that the greater asbestos exposure and retention an individual experiences, the greater the likelihood of his or her developing a disease. The question that is not addressed anywhere by Drs. Maddox and Laman is how they properly arrive at the conclusion that a dose response curve is applicable to the specific plaintiff before the court.

KNOWN DOSE REPONSE CURVES APPLICABLE TO HIGH DOSE EXPOSURE SHOULD NOT BE APPLIED TO LOW DOSE EXPOSURES WITHOUT THE SUPPORT OF GENERALLY ACCEPTED METHODOLOGIES.

Drs. Maddox and Laman do not rely, in any respect, upon any actual quantity or quality of exposure suffered by any specific plaintiff, but rather, conclude that if the evidence supports a single exposure, then causation can be opined and asserted. [12] Accordingly, Drs. Maddox and Laman are required to assert that an asbestos-related disease dose response curve applies even where there is a vanishingly small exposure. I have been unable to find, and I do not believe that Drs. Maddox or Laman, or any other witness or authority offered on behalf of the plaintiffs has offered any generally accepted methodology to support this proposition.

Asbestos exposure dose response rates have been studied and are the subject of a considerable volume of medical literature in the cases of high-dose exposures. In some trades, [13] much is known about the quantity and quality of asbestos fibers in the air during traditional work practices. Mathematical calculations can plausibly, if not ably, support reasonable assumptions about the amount of fibers inhaled by workers engaging in such traditional work practices. These values can then be correlated against the known incidence of a particular asbestos related disease within such worker populations. With appropriate adjustments for statistical and empirical error one can then, in turn, generate a reasonably reliable dose response curve. Such dose response curves have been generated for high dose exposures. I accept that dose response curves for high dose exposure do demonstrate an increased likelihood of disease with an increased dose of asbestos exposure. Dose response curves, based upon generally accepted scientific methodology, for "low dose" exposures, however, simply do not exist. [14]

Accordingly, in order to apply known dose response curves for high dose exposures to low dose exposures Maddox and Laman must "extrapolate down" from the premise that "exposure to large amounts of asbestos can cause disease" to the conclusion that "exposure to small amounts of asbestos can cause disease."

***7** The plaintiffs assert that Drs. Maddox and Laman are properly extrapolating from known facts and generally accepted scientific principles (i.e. known dose response curves for high dose asbestos exposure). Beginning with this generally accepted scientific principle, i.e. high dose exposure to asbestos may cause disease (and if high enough may be reasonably inferred to be the cause of a specific plaintiff's subsequently diagnosed asbestos related disease), Drs. Maddox and Laman attempt to "extrapolate down" reasoning that if high dose exposure is bad for you, then surely low dose exposure (indeed, no matter how low) must still be bad for you. In this regard, Drs. Maddox and Laman's argument and analysis encounters a simple logical error.

While it may be a valid assertion that: if high dose asbestos exposure is bad for you, then low dose asbestos exposure may *potentially* be bad for you; it is not a valid assertion that because high dose exposure to asbestos is bad for you, then low dose exposure to asbestos is, *in fact,* bad for you, or that a specific plaintiff's exposure at an unknown low dose exposure level, in fact, contributed to that plaintiff's asbestos-related disease.

The fallacy of the "extrapolation down" argument is plainly illustrated by common sense and common experience. Large amounts of alcohol can intoxicate, larger amounts can kill; a very small amount, however, can do neither. Large amounts of nitroglycerine or arsenic can injure, larger amounts can kill; small amounts, however, are medicinal. Great volumes of water may be harmful, greater volumes or an extended absence of water can be lethal; moderate amounts of water, however, are healthful. In short, the poison is in the dose. [15]

Plaintiffs cite *Trach v. Fellin,* 817 A.2d 1102 (Pa. Super 2003) to support the extrapolation analysis employed by Drs. Maddox and Laman. But *Trach* did not involve "extrapolation down;" rather it utilized "extrapolation up," or perhaps more precisely: "extrapolation away from the chemical norm."

Trach allowed a physician to opine that, where it was a generally accepted principle that exposure to known heightened dosages of Bendectin could cause certain birth defects, exposure to grossly higher levels of Bendectin could be reasonably anticipated to cause other adverse effects. *Trach* tells us what we understand common-sensibly, that when science knows that a certain deviation from a body's chemical norm causes harm, then a greater deviation from a body's chemical norm can be reasonably expected to cause increased harm (i.e. "extrapolation up"). What *Trach* does not say is that where it is known that a certain deviation from a body's chemical norm causes harm, a lesser deviation from a body's chemical norm can be similarly presumed to cause harm (i.e. "extrapolation down").

Employing an exaggerated example, while admittedly absurd, nonetheless, illustrates the point. If it is accepted by medical science that forcing an individual to drink 100 cups of water within an hour will have adverse effects upon his or her physical well-being, it is not unreasonable to offer medical opinion that forcing a person to drink 200 cups of water within one hour, will likewise cause ill effects; and moreover, may be predictably expected to cause even greater ill effects upon his or her well-being. However, it is not reasonable to "extrapolate down" from the known scientific fact that forcing a person to drink 100 cups of water within one hour can cause ill effects to the conclusion that forcing a person to drink 3 cups of water within an hour will cause ill effects upon his or her well-being. [16] The reasons for this conclusion are obvious, the human body may be able to tolerate or in some manner accommodate a small deviation from its chemical or biological norm, but greater deviations it cannot.

***8** Moreover, there may be limitations to the appropriate and responsible utilization of "extrapolation up" by experts in Pennsylvania courts. In *Vinitski v. Adler,* 2005 W.L.984497 (Pa.Super 2005), a memorandum decision, the Superior Court discussed the limitations of extrapolation under *Trach* stating:

> According to appellants such extrapolation [up] is allowed under Trach v. Fellin 817 A.2d 1102 (Pa.Super.2003) This is however a misreading of our opinion.
>
> The current appeal is a far cry from *Trach.* Here, Dr. Breggin wishes to start at the principle that Valium causes short-term and acute dementia and arrive, somehow, at the conclusion that long-term Valium use causes permanent frontal lobe brain damage. Yet, this is not logical; one cannot view the temporary effects a drug has on the brain and then leap to the conclusion that these temporary effects become permanent and, indeed much worse, with repeated exposures. This is not extrapolation, it is merely a biased guess.
>
> Thus, we agree with the trial judge: Dr. Breggin's methodologies do not proceed scientifically to his stated conclusion. As such, Frye prohibits his testimony as an expert.

Vinitski, 2005 WL 984497 (Pa.Super.), at page 3. [17]

As such, the rationale employed in *Vinitski* recognizes the limitations to extrapolation even where the extrapolation involves extrapolation away from the normal body conditions as opposed to "extrapolating downward" toward more normal body conditions.

Generally accepted scientific methodology may well establish that certain "high dose" asbestos exposure causes, or contributes to, a specific hypothetical plaintiff's disease, but the plaintiffs have not proffered any generally accepted methodology to support the contention that a single exposure or an otherwise vanishingly small exposure has, in fact, in any case, ever caused or contributed to any specific individual's disease, or even less so, that in this case such a small exposure did, in fact, contribute to this specific plaintiff's disease.

A SAFE LEVEL OF EXPOSURE?

Plaintiffs counsel has repeatedly argued and solicited testimony and admissions that "there is no safe level of exposure to asbestos ." This assertion, while in one manner of speaking not necessarily inaccurate, implicitly suggests a placement of the burden of proof with the wrong party and tends to misdirect the fundamental inquiry.

First, the plaintiffs are required to demonstrate that generally accepted methodology within the relevant scientific field supports their proffered expert opinions that low dose asbestos exposure causes disease generally and in the specific plaintiff before the court. The defendants do not maintain a

burden of proving the contrary or that "there is a safe level of asbestos exposure".

Second, when considered in context, all expert witnesses who have agreed with the statement that "there is no safe level of asbestos exposure" have done so to the extent that they agree that there is no *known* safe level of exposure. This is an important distinction. There may, or there may not, be an actual safe level of asbestos exposure. The critical point for my purpose is that, at present, whether there is (or is not) a safe level of asbestos exposure is currently unknown utilizing generally accepted scientific methodology.

***9** So, while it may be accurate that there is no competent evidence in this record that supports the position that medical science has confirmed, or can support a reasonable inference that, there exists a safe "low dose" level of exposure to asbestos; [18] it is likewise accurate that there is no competent evidence in this record that supports the contention that medical science is able to confirm, or otherwise support a reasonable inference, that each and every exposure to asbestos contributes to a subsequently diagnosed asbestos-related disease. Finally, there is no competent evidence in this record that supports the conclusion that the quality and quantity of the exposure[s] that a jury might reasonably find or infer that the plaintiff[s] in this case experienced while performing occupational duties with and around friction products, caused or in any way contributed to the development or progression of this plaintiff's asbestos-related disease. [19]

Parenthetically, some members of the Superior Court have offered guidance (albeit in dicta) on the question of whether a vanishingly small exposure may be reasonably found to be a substantial contributing factor to a plaintiff's illness. In an evenly split *en banc* decision, the Superior Court's Opinion in Support of Affirmance in *Summers v. Certainteed Corporation, et al.,* 886 A.2d 240, stated:

> Just because a hired expert makes a legal conclusion does not mean that a trial judge has to adopt it, if it is not supported by the record and is devoid of common sense. For example, Dr. Gelfand used the prhrase, "each and every exposure to asbestos has been a substantial contributing factor to the abnormalities noted." However, suppose an expert said, "that if one took a bucket of water and dumped it in the ocean, that was a 'substantial contributing factor' to the size of the ocean. Dr. Gelfand's statement saying every breath is a "substantial contributing factor" is not accurate. If someone walks past a mechanic changing brakes, he or she is exposed to asbestos. If that person worked for thirty years at an asbestos factory making lagging, it can hardly be said that the one whiff of the asbestos from the brakes is a "substantial" factor in causing disease.

Summers, Opinion in Support of Affirmance, 886 A.2d at 244.

Generally accepted scientific methodology is not able to demonstrate what effect low dose exposures have upon the body. While there plainly exists anecdotal suspicion that each and every exposure to asbestos fibers *might,* potentially, possibly, contribute to an asbestos-related disease; such anecdotal suspicions are, in my judgment, a far cry from the quantum and quality of evidence necessary to present expert opinion testimony to juries in Pennsylvania.

PROOF OF "INCREASED RISK"
DOES NOT ESTABLISH CAUSATION

It is black-letter law in Pennsylvania that causation in a product liability case requires actual causation and injury, not simply the increase of risk of injury to the plaintiff. In E*ckenrod v. GAF Corp.,* 544 A.2d 50 (Pa.Super 1988), the Superior Court stated: "In order for liability to attach in a product liability action, Plaintiff must establish that the injuries were caused by a product of the particular manufacturer or supplier.... Summary Judgment is proper when the Plaintiff has failed to establish that the Defendants' products were the cause of Plaintiff's injury." E*ckenrod,* at 52.

***10** In order to be admissible, an expert's opinion, attributing an illness to a specific cause, must be made with the requisite degree of certainty, as stated in *McMahon v. Young,* 276 A.2d 534 (Pa.1971):

> (T)he expert has to testify, not that the condition of claimant might have, or even probably did, come from the accident, but in his professional opinion the result in question came from the cause alleged. A less direct expression of opinion falls below the required standard of proof, and does not constitute legally competent evidence. (citing cases).
>
> The issue is not merely one of semantics. There is a logical reason for the rule. The opinion of a medical expert is evidence. If the fact finder chooses to believe it, he can find as a fact what the expert gave as an

> opinion. For a fact finder to award damages for a particular condition to a Plaintiff, it must find as a fact that that condition was legally caused by the Defendant's conduct. Here, the only evidence offered was it was 'probably' caused, and that is not good enough. Perhaps in the world of medicine nothing is absolutely certain. Nevertheless, doctors must make decisions in their own profession every day based on their own expert opinions. Physicians must understand that it is the intent of our law that if the Plaintiff's medical expert cannot form an opinion with sufficient certainty so as to make a medical judgment, there is nothing on the record with which a jury can make a decision with sufficient certainty so as to make a legal judgment.

McMahon, 276 A.2d at 535, citing *Menarde v. Philadelphia Trans. Company,* 103 A.2d 681 (Pa.1954).

In *Checchio v. Frankford Hospital-Torresdale Division,* 717 A.2d 1058(Pa.Super.1998) (implicitly overruled on other grounds), the Superior Court addressed the general acceptance of the methodologies proffered to support expert opinions causally attributing a two-year-old's cognitive deficits to negligent medical treatment rendered in response to respiratory distress immediately following the child's birth. The *Checchio* court stated:

> The crux of [Plaintiff's] argument and the logical construct on which their case is grounded begins with the major premise that a lack of oxygen and blood flow to the brain can cause neurologic damage. Daniel suffers neurologic damage, the argument proceeds therefore the damage must have been caused by oxygen deprivation. The corollary to this conclusion is that 'the damage may manifest itself in a severe mental retardation, developmental delay, and autistic like behavior exhibited by Daniel Checchio.'

Checchio, 717 A.2d at 1060.*Checchio* goes on to state:

> [Plaintiffs] argue that these opinions are sufficient to satisfy *Frye* because '[i]t is a well established fact in the medical community that a lack of oxygen to the brain will eventually cause hypoxia and if severe and prolonged enough it will result in acidosis and eventual death of the brain tissue.' ... While this may well be true, it does not explain whether the specific condition from which Daniel suffers is the result of brain tissue death or some other cause, ...
>
> ***11** Nor, again, even accepting the validity of [Plaintiff's] major premise, does it serve to connect the autistic tendencies with the brain injury. There is no testimony, and no evidence of any other sort, to the effect that such tendencies always occur in conjunction with hypoxic brain damage, or indeed with mental retardation. Appellants themselves assert somewhat tentatively that the neurologic dysfunction allegedly caused by the putative hypoxia may cause the condition exhibited by Daniel ...; they paraphrase their own experts, who are actually more positive in their assessment, as asserting that 'a lack of oxygen can cause brain damage' (emphasis added). No authority, statistical or otherwise is offered on these points.

Checchio, 717 A.2d at 1061 (emphasis in original). As in *Checchio,* so it is in this case that, the Plaintiff's experts do not offer support or methodology other than their subjective belief that each and every breath of asbestos causes or substantially contributes to the disease process suffered by the Plaintiff.

While it is true that Drs. Maddox and Laman do not materially equivocate with regard to the certainty of their professional opinion, they offer not a shred of independent corroboration of their opinion that each and every fiber causes or contributes to a Plaintiff's disease process. To the extent that it is suggested that these wholly unsupported assertions might be more fairly interpreted as simply a statement that each and every inhalation of asbestos fibers *increase the risk* or *probability* of the Plaintiff suffering from asbestos-related disease or every inhalation might *possibly* cause or contribute to the Plaintiff's disease, such opinions proffered in support of the causation prong of the Plaintiff's claim are simply not admissible under the principles of *Menarde,* and *McMahon.*

THE EXISTENCE OF THE DISEASE COUPLED WITH EXPOSURE HISTORY ALONE SHOULD NOT BE PERMITTED TO ESTABLISH CASUATION WITHOUT GENERALLY ACCEPTED METHODOLOGIES TO SUPPORT THE ATTRIBUTION.

Plaintiffs, at times, seem to implicitly assert that the mere existence of the asbestos-related disease [20] coupled with the allegation (or proof) of exposure to the defendant's product can support a finding of causation. This argument is without

merit. Demonstration of the lack of merit is dependent upon the disease process involved.

In the case of mesothelioma, while I recognize that there exists a consensus in the scientific community that mesothelioma *may* be caused by asbestos exposure levels far less than those necessary to cause asbestosis or pleural fibrosis, [21] both Drs. Maddox and Laman (as well as all of the defendants' witnesses) recognize that the probability of development of mesothelioma is particular to the individual. They recognize that many individuals with the same level of exposure (whether below, at, or above normal background levels [22]) will not develop mesothelioma. Not surprisingly, some people exposed to the same level of asbestos will develop asbestos-caused mesothelioma, while others will not; and others may not develop asbestos-caused mesothelioma at even much greater levels of exposure.

***12** All of the witnesses, including Drs. Maddox and Laman acknowledge that a certain percentage of mesotheliomas are idiopathic. The phrase "idiopathic" is intended to describe diseases that develop without a known, or attributable, cause. Presumptively, an idiopathic mesothelioma can develop, whatever its cause, in an individual with no asbestos exposure, normal background level asbestos exposure, or greatly heightened asbestos exposure. Estimates of the incidence of idiopathic mesothelioma range from 6% to 20 % of all reported mesotheliomas. [23] Presumably, because of the possibility of an idiopathic mesothelioma, which can occur even in the absence of asbestos exposure, both Drs. Maddox and Laman reserve their attribution of mesothelioma to asbestos exposure only where there is evidence that the plaintiff has experienced an increased exposure to asbestos over the normal background levels. [24]

But Drs. Maddox and Laman still never satisfactorily answer the question of how they can distinguish an idiopathic mesothelioma that would have occurred in a given plaintiff regardless of his lifetime asbestos exposure (whether non-existent, average or high) from a purportedly "asbestos-caused mesothelioma" in an individual with, at best, modestly increased lifetime asbestos exposure. [25] Moreover, even where it is conceded that a mesothelioma was caused by asbestos exposure generally, neither Maddox nor Laman ever addresses how it is that they can determine that it was exposure to a specific defendant's friction product that caused a plaintiff's mesothelioma and not some other asbestos exposure that independently caused the mesothelioma.

Where the plaintiff suffers from asbestosis or some form of pleural fibrosis, the experts generally acknowledge that these disease processes are caused by exposure to asbestos generally. What remains contested is whether or not the exposure to a specific friction product was the cause of (or contributor to) these asbestos-caused diseases. Because, as discussed earlier, certain unknown factors related to "low dose" exposures exist within the realm of known medical science, including whether "low dose" exposures contribute at all to asbestosis or pleural fibrosis and, if so, to what degree and under what conditions, Drs. Maddox and Laman do not offer an opinion based upon generally accepted methodologies that the plaintiff's low dose exposures to a specific defendant's friction products was a substantial contributing factor to his or her asbestos-related disease.

PLAINTIFFS DO NOT RELY UPON BIOLOGICAL FINDINGS TO SUPPORT THEIR CLAIMS AGAINST ANY PARTICULAR DEFENDANT

Numerous studies have been conducted that determine the amount of fibers that have been retained within different biological structures of individuals with known asbestos-related exposure and/or disease. Accordingly, reliable information is available to the relevant scientific communities regarding the quantity and quality of fibers retained in biological structures of individuals exposed to asbestos who subsequently develop asbestos related disease. In some instances, where statistically significant high fiber loads are recognized, a medical consensus (or at least generally accepted methodologies) exist to support an opinion causally attributing an individual's asbestos-related disease to asbestos exposure. [26] There are no "fiber load" findings relied upon by the plaintiffs experts in this case.

PLAINTIFFS DO NOT RELY UPON QUANTITATIVE EVIDENCE OF OCCUPATIONAL HIGH DOSE EXPOSURE TO SUPPORT THEIR CLAIMS AGAINST ANY PARTICULAR DEFENDANT

***13** Neither Dr. Maddox, nor Dr. Laman attempts to meaningfully quantify the actual or even approximate amount of plaintiff's occupational, or other, asbestos exposure in this case. Nor do they attempt to meaningfully quantify the exposure directly attributable to a specific defendant's friction

product. Moreover, nowhere do they attempt to delineate a threshold exposure, or even a potential range for a threshold exposure, (i.e. over which they would attribute a specific exposure and under which they would not attribute a specific exposure as a cause of an asbestos related disease), other than to simply indicate that if there was a single exposure to a defendant's asbestos containing product, then the plaintiff's disease can be causally attributed to that exposure. Drs. Maddox and Laman do not, however, offer any methodology other than those addressed above to support that conclusion.

A DIFFERENT RESULT MAY BE WARRANTED WHERE PLAINTIFFS EXPERTS' OPINIONS ARE BASED UPON GENERALLY ACCEPTED METHODOLOGIES UTILIZING BIOLOGICAL FINDINGS OR OTHER QUANTITATIVE EVIDENCE OF ACTUAL HIGH DOSE EXPOSURE.

Armed with quantitative information from biological samples reflecting statistically significant higher fiber loads within the plaintiff's biological structures, or quantitative evidence of statistically significant actual high dose asbestos exposure the scientific community can, in some instances, utilize generally accepted methodologies to causally attribute an asbestos-related disease to a known quantity of high dose asbestos exposure. From this data, the relevant scientific communities have developed some understanding and appreciation for the fact that given a certain quantity and quality of exposure to a particular asbestos-containing product, a fair inference can be drawn that that product contributed to an asbestos-related disease. Precisely what quality and quantity of exposure is necessary is the subject of honest debate within the relevant scientific communities.

Where the debate falls silent, however, is in the area of low-dose exposures or where, as here, there exists no quantitative evidence of either actual occupational exposure or biological samples from which causal attribution can be reasonably inferred. Frankly, because such low-dose exposures do not, as strongly, correlate to asbestos-related disease, there is less, and in some instances, no information available to scientific inquirers regarding whether low-level asbestos exposure, in fact, contributes to an asbestos related disease.

Accordingly, while Drs. Maddox and Laman's opinions regarding medical causation of asbestos-related diseases in large-dose scenarios are supported by the medical literature (and, in fact, are perhaps a consensus opinion among medical experts), what is neither equally the subject of a medical consensus, nor even supportable by generally accepted methodology is the opinion that low-dose asbestos exposures are causative of asbestos-related diseases generally, let alone in this specific plaintiff. There is no medical authority or generally accepted methodology that would support the conclusion that low-dose exposures cause asbestos-related disease generally, let alone the rather extraordinary assertions by Drs. Maddox and Laman that “each and every exposure” substantially contributed to this specific plaintiff's disease process. It is in this regard, that this court ultimately concludes, that Dr. Maddox's and Dr. Laman's methodology is fundamentally flawed and not generally accepted by the relevant scientific community.

THIS RULING IS BASED UPON INADEQUACIES IN THE PLAINTIFF'S EXPERTS' METHODOLOGIES, NOT UPON THE PROFFERED MERIT OF DEFENDANTS' EPIDEMIOLOGICAL STUDIES, OR OTHER EXPLANATIONS FOR WHY “FRICTION PRODUCTS ARE DIFFERENT.”

***14** Much of the *Frye* hearing addressed the defendants' argument that there exist numerous plausible explanations for why exposure to friction products (i.e. brakes and clutches generally) does not, in fact, contribute to the development of asbestos-related disease. These arguments centered upon the assertions that 1) fibers shed from friction products are chemically altered during use so as to render them biologically inactive and harmless, 2) that those fibers that are not so altered are too small to contribute to the disease process, and 3) that traditional work place asbestos exposures for individuals who work with friction products, (i.e. brake/auto mechanics) are not adequate levels of exposure to meaningfully contribute to asbestos-related disease. Moreover, the defendants rely upon proffered epidemiological studies to statistically support their contention that occupational exposure to friction products does not substantially contribute to asbestos-related disease.

While this evidence is academically interesting and satisfies a certain degree of curiosity as to why friction products might appear to cause (or not cause) disease differently than other asbestos-related products, this evidence does not, in my judgment materially support the defendants' Frye challenge. Specifically, I do not hold that the expert opinions explaining “why friction products are different” or the epidemiological evidence offered by the defendants in this case, in any

manner, "trumps" the plaintiff's evidence,[27] or that the Plaintiffs are required to proffer epidemiological evidence in support of their medical causation opinion.

The defendants assert that where an opinion (such as Drs. Maddox and Laman's) relies fundamentally upon case reports and extrapolation from known facts, it can be properly argued that the opinion is no longer generally accepted by the relevant scientific community where, as a function of the advance of scientific knowledge, stronger, more conclusive, evidence is now available in the form of epidemiological studies.[28] Frankly, the attempt to focus upon the defendants' epidemiological evidence and other explanations for "why friction products are different" is simply improper.[29] The focus of this court should be, and has been, upon what methodologies were utilized by Drs. Maddox and Laman, and whether those methodologies support the conclusions proffered by Drs. Maddox and Laman and whether those methodologies are, in fact, generally accepted within the relevant scientific fields.

SMALLS AND *ANDALARO*

Of particular concern to me is that my ruling on this Frye challenge may appear, facially, to be at odds with the ruling of the Superior Court in *Smalls v. Pittsburgh Coming, et al.* 2004 PA Super 31, 843 A.2d 410 (Pa.Super.2004). Because of this concern, I set forth the relevant language of *Smalls* in its entirety:

> Next, Appellant asserts that the trial court erred in allowing Dr. Richard Katz, Appellees' expert, to testify as follows: "Each and every breath of asbestos fibers is [a] significant and substantial contributing factor to the asbestos related disease that Mr. Smalls has." N.T. Trial, 12/4/01, at 32. Appellant argues that the opinion was inadmissible because it had no basis in fact nor general acceptance in the scientific community. We disagree.
>
> ***15** Again, we observe that the admission or exclusion of evidence is within the discretion of the trial court, and it will not be reversed absent a manifest abuse of that discretion. *Eichman,supra.* As we previously have held that this type of opinion evidence is not only admissible, it is sufficient to demonstrate a *prima facie* case of liability against an asbestos manufacturer if believed by the fact finder, see *Junge v. Garlock Inc.,* 427 Pa.Super 592, 629 A.2d 1027 (1993), the trial court's decision to admit the statement was not tantamount to an abuse of discretion. Moreover, we observe that Dr. Katz is certified by the American Board of Medical Specialties in pulmonary disease, and his experience and expertise is sufficient to testify about the relationship between breathing asbestos and the development of asbestos-related diseases.

Smalls, 843 A.2d at 414. I have considered *Smalls,* and I have earnestly attempted to comply with any mandates that may be set forth within it.

Initially it should be noted that it does not appear from the Superior Court's decision in the *Smalls* case that the trial court actually conducted a *Frye* hearing. Accordingly, the trial court may have simply concluded that the proffered expert testimony was not novel, and a *Frye* hearing not necessary. Moreover, although the Superior Court does not disapprove of the trial court's failure to sustain a *Frye* objection to the testimony by plaintiff's expert that: "Each and every breath of asbestos fibers is [a] significant and substantial contributing factor to the asbestos-related disease that [plaintiff] has." The Superior Court provides no analysis of why such an expert opinion is, in fact, generally accepted in the relevant scientific community, and cites no facts of record that could plausibly support such a conclusion.

To the extent the Superior Court provides an analysis, it does so in the following paragraph where it indicates that a trial court's decision to exclude evidence is within the trial court's discretion, and that opinion evidence of the type offered in *Smalls* has been held to be admissible and sufficient to demonstrate a *prima facia* case of liability. The Superior Court cites *Junge v. Garlock, Inc.* in support of this final proposition, but fails to note that the *Junge* case did not involve a Frye challenge.

Accordingly, while the specific language set forth in *Smalls* is heavily relied upon by the plaintiffs; and it is true that that language, standing alone, could conceivably support the assertion that the Superior Court has explicitly taken judicial notice that there is general acceptance in the scientific community that: "Each and every breath of asbestos fibers is [a] significant and substantial contributing factor to the asbestos-related disease that the [plaintiff] has," this court simply cannot bring itself to conclude that that was the intent of the Superior Court in *Smalls.*

***16** First, and most significantly, the focus of a Frye challenge in Pennsylvania is not on the general acceptance of the *opinion* proffered, but rather on the general acceptance

of the *methodology* underlying the opinion. No consideration of the methodology supporting the proffered opinion in *Smalls* was ever undertaken. If the *Smalls* holding was intended as suggested by the plaintiffs (i.e. a declaration that Pennsylvania courts have recognized that the opinion: "Each and every breath of asbestos fibers is [a] significant and substantial contributing factor to the asbestos-related disease that the [plaintiff] has" enjoys general acceptance within the relevant scientific field) then the Superior Court's analysis in *Smalls* would have improperly focused on the irrelevant question of whether there existed general acceptance of the *opinion* of the expert and not on the proper inquiry of whether there is general acceptance of the *methodology* underlying that opinion. I do not conclude that the *Smalls* court intended to so focus its analysis and ruling.

Second, if interpreted as suggested by plaintiffs, *Smalls* would constitute a judicial decree potentially usurping the collective expertise of the medical/scientific community regardless of the actual general acceptance (or lack thereof), within the relevant scientific field, of whatever methodologies, (whether sound or wholly preposterous) that may have been proffered in support of the expert's opinion in *Smalls,* without a moment's substantive consideration. I plainly cannot conclude that this was the intent of the *Smalls* Court.

Rather, I interpret *Smalls* to simply indicate that where a trial court, within its discretion, does not determine that proffered expert testimony is novel, and then concludes that such testimony is admissible, the Superior Court will not disturb such a ruling, because such a ruling does not constitute a manifest abuse of the trial court's discretion.

Additionally, plaintiffs assert that the Superior Court's decision in *Andaloro v. Armstrong World Industries, Inc.* 799 A.2d 71 89 (Pa.Super.2002) similarly supports the position that Pennsylvania courts have accepted that "each and every breath of asbestos fibers is a significant and substantial contributing factor to the asbestos-related disease [that a plaintiff has developed]" and that, in fact, no further proof of causation of injury is required than to demonstrate inhalation of asbestos fibers by an individual with an asbestos related disease. However, the *Andaloro* opinion expressly concludes that the defendant "failed to preserve for appellate review its claim that the causation theory advanced by plaintiff's experts was not generally accepted in the scientific community." Because the defendant did not preserve its *Frye* objection, the Superior Court's language related to the "Frye" issue in *Andaloro* is dicta and not binding on this court. However, because *Andaloro* directly addresses, (albeit in dicta), an issue presented to, and preserved before this court, I have set forth the relevant language from *Andaloro* below:

> ***17** [Defendant's] assertion is derived from the premise that quantification of the levels of asbestos exposure a plaintiff suffered is a prerequisite to a determination of causation and hence, liability. [Defendant] provides no authority for such a premise, nor are we aware of any. In point of fact, Pennsylvania law provides that causation of asbestos-related injuries is shown upon proof that the plaintiff inhaled *some* fibers from the products of the defendant manufacture ...
>
> > Our case law includes no requirement that a plaintiff in an asbestos case prove through [expert testimony] *how many asbestos fibers are contained in the dust emissions from a particular asbestos-containing product.*
>
> Similarly, the plaintiff need not demonstrate the specific links of fibers contained in the manufacturers product, the length of fibers he inhaled, or the overall concentration of fibers in the air ... Because these elements are not legally necessary to a determination of causation, an expert's inability to testify about them does not render his testimony incompetent on the issues of causation and liability.

Andaloro, 799 A.2d at 85-86 (emphasis in original) citing *Junge v. Garlock,* 629 A.2d 1027, 1029 (Pa.Super.1993).

The *Andaloro* decision might appear upon immediate review to hold that causation in an asbestos case is fully proven upon proof that the Plaintiff inhaled some fibers from the Defendants' product. However, a more reasoned interpretation of the language in *Andaloro* supports the conclusion that *Andaloro* merely recognizes the physical fact that in order for asbestos fibers to cause disease processes within the human body, they must first be inhaled. *Andaloro* does not, in any material manner, and certainly in no explicit respect, appear to challenge the generally applicable legal principle requiring proof of actual injury in a product liability case. Clearly, the more fundamental focus of the *Andaloro* court was that a Plaintiff need not prove the specific quantity of fibers inhaled, or the specific character or quality (including specifically the fiber length) of the fibers inhaled, but rather simply must establish that sufficient fibers were inhaled to have caused the Plaintiff's injuries. But again proof of actual injury remains a requirement.

Further guidance regarding the applicability of the *Smalls* and *Andaloro* cases is found in the Superior Court's decision in *Rafter v. Raymark, Industries,* 632 A.2d 897 (Pa.Super 1993): where the court stated as follows:

> Appellant argues that [the court's jury instructions] led the jury to believe that if they found that appellees inhaled asbestos, then [the jury] must also conclude that asbestos was a substantial cause of [the Plaintiffs'] lung and throat cancer. We disagree.... In the instant action, the trial court never stated that inhalation of asbestos was sufficient but, rather, stated that it was necessary to establish that asbestos exposure was a substantial factor in causing [Plaintiffs'] injuries. Moreover, the trial court's instruction clearly provided that appellees were required to show that they have been injured by asbestos exposure, and that this exposure was a substantial contributing factor to their injuries.... After reviewing the instruction in its entirety, we find no abuse of discretion or error of law regarding the trial court's charge on causation.

***18** *Rafter,* 632 A.2d at 901-902 (citations omitted).

As demonstrated by the Court's analysis in *Rafter,* it is appropriate to conclude that the Superior Court does not approve of the notion that the mere inhalation of fibers is presumptively sufficient to establish causation in an asbestos case. Rather, proof of causation of the actual injury is necessary.

CONCLUSION

For the reasons set forth above, I entered the Order of February 27, 2006, precluding the plaintiff's experts from offering opinion testimony causally attributing the plaintiff's asbestos related disease to exposure to any specific defendant's friction products.

Footnotes

1 Resolution of this Motion was assigned to me by Order of Court of the Administrative Judge of the Civil Division of Allegheny County.

2 It is not my intent that the phrase “friction product manufacturer” be interpreted as a strict term of limitation, but, generally speaking, I intend by use of the phrase to identify brake and clutch manufacturers.

3 The cases identified by counsel were: *Simikian v. [Asbestos Defendants],* GD 05-004662; *Bahneman v. [Asbestos Defendants]* GD 04-010451; *Cline v. [Asbestos Defendants],* GD05-010028; and *Vogelsberger v. [Asbestos Defendants]* GD 02-18135. Each has been incorporated by reference at Administrative Docket No. A.D. 03-000319.

4 By “non-case-specific” expert reports, I intend to describe expert reports specifically filed in each case, but which did not rely upon specific factual circumstances involved in the case. For instance, in each instance, neither Dr. Maddox nor Dr. Laman relied upon specific data regarding the quality or quantity of the specific plaintiff's alleged exposure to any of the specific defendant's product, or to friction products generally; but rather the reports essentially theorized that if the evidence offered at trial established any work around or exposure to friction products then a finding of proximate causation may be supported for the reasons proffered in the expert reports. These reports are substantially identical to reports prepared by Drs. Maddox and Laman previously filed on behalf of numerous asbestos plaintiffs.

5 I do not mean to unfairly disparage the honestly held beliefs of Drs. Maddox and Laman. I maintain no doubt that the doctors' opinions are rooted in each doctor's abundant knowledge of the best evidence currently available to science regarding asbestos exposure generally. Indeed, I will be among those least surprised if, some day, generally accepted scientific methodology validates some or all of Drs. Maddox and Laman's opinions in this case. Their opinions enjoy a certain commonsensical appeal, and are not, in any specific respect, disproved by medical science. This, however, is not the standard for admissibility of expert opinion in Pennsylvania. In the end, the doctors' opinions are nothing more than their current “best guesses,” unverified by generally accepted methodology. This kind of expert opinion is simply not admissible in Pennsylvania courts.

6 I borrow greatly from, and am indebted to, the excellent analysis set forth in Judge Allen's trial court opinion in the case of *Vinitski v. Adler,* 69 Pa. D & C 4th 78. (Pa.Com.Pl., Phila., 2004), affirmed by memorandum opinion 2005 WL 984497 (Pa.Super.2005).

7 The facts set forth in this section are not materially contested by the parties.

8 I specifically acknowledge and recognize as binding, the ruling of the Superior Court in *Trach v. Fellin,* 817 A.2d 1102 (Pa. Super 2003), that epidemiological studies are not necessary to demonstrate that exposure to an agent was the medical cause of a disease. Still, *Trach* does not alter the fundamental principle that proof of actual causation remains required.

9 I do not take issue with this predicate opinion regarding general causation, for purposes of this *Frye* ruling.

10 Coffee drinking coffee strongly correlates with smoking; which, in turn, strongly associates coffee drinking with lung cancer.

11 My use of the phrase "high dose" is not intended to imply or suggest any particular quantitative value of dosage but rather to simply distinguish between the "low dose" of a single exposure, or otherwise vanishingly small exposure that is opined to be a proximate cause of a specific plaintiff's disease by Drs. Maddox and Laman and certain known higher dose exposures that can, and have been, adequately associated with asbestos related disease based upon generally accepted scientific methodologies.

12 In fairness, Maddox and Laman, at times, seem to suggest that, given any *reasonable* length of employment as an auto/brake repairman, a specific plaintiff's actual exposure should be adequate to allow Maddox and Laman to opine the applicability of a dose response curve. Nowhere, however, do they even remotely attempt to quantify the actual exposure that they believe would be required, or support how they arrive at a quantitative value for a specific plaintiff's exposure. As such, when subjected to even modest scientific rigor their low dose causation "methodology" is either not stated or fails, unless they can support the contention that each and every exposure contributes to the disease process.

13 In particular, the scientific literature establishes some understanding of applicable dose response curves for exposures typically experienced by high exposure trades including asbestos miners, asbestos insulators, and ship workers, among others.

14 Parenthetically, it has been asserted that dose response curves for low dose exposure do not exist, in part, because of an absence of reliable information regarding actual inhalation and retention of asbestos fibers in low exposure settings and, in part, because of a statistically modest, (or non-existent) increase of the occurrence of asbestos related disease at low exposure levels. Whatever the reason for their non-existence, *why* they do not exist is not determinative of my ruling. *The fact that they do not exist,* and thus, cannot be relied upon by Drs. Maddox and Laman is the important point.

15 I do not intend to assert that asbestos is, in fact, ever medicinal or benign in any quantity, but only that the "extrapolation down" assertion and argument relied upon by Maddox and Laman can not logically establish, or give rise to a reasonable inference, to the contrary.

16 I recognize that this example does not constitute an exact parallel to the position of the plaintiff's experts, but it illustrates the point effectively enough.

17 In citing to *Vinitski,* a memorandum decision, I am cognizant of *Internal Operating Procedure of the Superior Court,* Rule 444B, implementing Pa.R.A.P. 3501-3517, and Superior Court *Notice to the Bar,* 598 A.2d 1324. My citation is not intended to rely upon *Vinitski* as precedential authority but rather only to illustrate (consistent with *Melendez v. Pennsylvania Assigned Claims Plan,* 557 A.2d 767 (1989) holding that because "the authority relied upon in [the cited memorandum decision] applie[d] to the instant case, ... the trial court's conclusion was correct,") that my proffered interpretation of *Trach* is reasonable. If it is determined that I have improperly relied upon *Vinitski* in this respect, I note that my ruling is only buttressed, and not exclusively based upon, that reliance and, thus, if improper the reliance may be deemed harmless. *Major v. Major,* 518 A.2d 1267 (1986).

18 Of course, I recognize, and accept, that generally accepted scientific methodology supports the opinion that sufficiently "high dose" (comparatively speaking) exposures of asbestos are known to be dangerous. Additionally, I understand, and accept, that there is no competent evidence in this record that supports the position that medical science has confirmed, or can support a reasonable inference that, there does exist a safe "low dose" level of exposure to asbestos. Accordingly, I am aware that there may, in fact, be no safe level of exposure. I, and everyone else, simply do not know.

19 This is not to say that I know that the plaintiff's occupational exposure to the defendant's friction products (if proven) did not, in fact, contribute to plaintiff's disease-it very well may have. But, based upon this record, (subject to the limitations of scientific knowledge, as it is) that is about as much as I, or anyone, can say on the subject-"it *may* have." "It may have" is not a sufficient basis for a jury to find, (or to reasonably infer), that it *did.* To allow a jury to find, (or infer), that "it *did"* where the evidence supports, at best, the conclusion, or inference, that "it *may* have" simply invites the jury to guess; and that, the jury may not do.

20 I intend to reference specifically mesothelioma, asbestosis, and forms of pleural fibrosis.

21 There appears to be a consensus that mesothelioma can also be caused by other known factors, such as high levels of radiation exposure, that are not implicated in these cases.

22 Asbestos is naturally occurring in the environment, and thus we all unavoidably experience a (particularly insignificant, but) certain level of asbestos exposure.

23 I have considered the suggestion that because Maddox and Laman are competent to report that medical consensus has established that between 80% and 94% of all mesotheliomas are caused by exposure to asbestos that they should, therefore, be permitted to offer the opinion, and the jury should be capable of finding, that it is more likely (in fact 80%-94% more likely than not) that this plaintiff's mesothelioma was caused by asbestos exposure. If I were to permit Drs. Maddox and Laman to offer such an opinion, I would be allowing a new method of proof of causation of injury in Pennsylvania. I suspect that such a method is not endorsed because it confuses proof of actual injury to a specific plaintiff with proof of an increased risk of harm not necessarily applicable to any specific plaintiff. In practice it would allow for absurd results. For instance: to permit such a theory to be presented to a jury would

be tantamount to permitting a state trooper to offer an opinion that speeding caused a specific one-car accident based solely upon his knowledge that 80% of all other one-car accidents on Pennsylvania highways are caused by speeding. To permit such testimony to be presented to a jury as expert testimony in support of a theory of causation would invite sheer speculation to replace reason and logic in support of a medical causation finding.

24 Interestingly their opinion in this regard does not appear to take into account their recognition that "normal" background levels are different in different parts of this country, and around the world, as a function of local geography, geology, and industrial development.

25 Allowing that plaintiff[s] in this case are "individual[s] with, at best, modestly increased lifetime asbestos exposure" gives the plaintiff's experts the benefit of the doubt, as they rely on no actual data regarding the plaintiff[s] actual lifetime exposure, but merely allow that if there was any exposure to the defendant's product, then causation can be found.

26 While I need not, and do not, reach the issue squarely, there exists a secondary question as to whether causal attribution to exposure to a specific product may be made based solely upon biological "fiber load" findings, particularly where there is sufficient evidence of other asbestos exposure, and there exists no meaningful generally accepted methodology to determine the actual, or likely, fiber source detected in pathological samples.

27 Additionally, it should be noted that I am cognizant of the concerns raised by members of the Supreme Court in *Blum v. Mergenthal* regarding the manufacturing of scientific consensus by corporate interests. It has been insinuated at various times during these *Frye* proceedings that the epidemiological evidence proffered by the defendants is this type of manufactured cannon fodder. While I can discern no such ulterior or improper motives on behalf of the researchers who conducted the studies that constitute the substantive basis for the epidemiological evidence presented by the defendants in this *Frye* hearing, it remains important to note that I do not ultimately rely upon the epidemiological evidence to support my ruling. As such, the legitimate concern of the Supreme Court regarding such manufactured evidence, while perhaps proper in some cases, would be misplaced in this case

28 I reject this assertion based upon my interpretation of the spirit of *Trach'* s clear directive that plaintiffs are not required to advance epidemiological evidence to prove causation. If I am mistaken in this regard, guidance from the appellate courts regarding the appropriate, required, or allowable consideration of epidemiological evidence countering the plaintiff's proffered methodologies within the context of a *Frye* challenge would be welcomed.

29 I note that in the face of ever-increasing scientific knowledge, the appellate courts of Pennsylvania may, someday, choose to revisit the question of whether epidemiological evidence is necessary to establish causation in toxic tort cases. Until that day, however, this court will not entertain the argument that a Plaintiff must advance epidemiological evidence to prove causation.

EXHIBIT 5

2007 WL 712049 (Pa.Com.Pl.) (Trial Order)
Court of Common Pleas of Pennsylvania.
Indiana County

Katherine M. BASILE, Executrix of the Estate of Fred Dalbo, Sr., deceased
and Viola Imogene Coen Dalbo, his wife, in Her own right, Plaintiffs,
v.
AMERICAN HONDA MOTOR COMPANY, INC. Et. al., Defendants.

No. 11484 CD 2005.
February 22, 2007.

Opinion and Order of Court

Gregory A. Olson, Judge.

Before the Court is Caterpillar Inc.'s Motion to Exclude Plaintiffs' expert testimony that relies upon novel scientific evidence, commonly referred to as a *Frye* motion. For those reasons set forth in the Opinion below, Caterpillar's motion is granted in part.

FACTUAL AND PROCEDURAL HISTORY

This is a toxic tort case. Plaintiff claims her Decedent, Fred Dalbo, Sr., died of mesothelioma he contracted as a result of exposure to asbestos fibers. Plaintiff has sued a number of defendants on the theory that Decedent's work-related exposure to asbestos fibers shed by one or more of defendants' products was a legal cause of Decedents' death.

In the course of pretrial discovery, Plaintiffs identified as potential expert witnesses two doctors, Christopher Faber and John C. Maddox.1 Each of these experts, by methodologies of case study and downward dose-effect extrapolation, opines that because asbestos exposure and consequent mesothelioma are cumulative processes, any asbestos exposure from any Defendant's product, whatever its nature and regardless of quantity or quality of exposure, is a legal cause of Decedent's mesothelioma.

By Order entered August 15, 2006, the Court per Pa. R.C.P. No. 207.1 directed that all *Frye* motions must comply with the Rule and set a filing and service deadline of January 15, 2007, with hearing on all *Frye* motions to occur on February 2, 2007, at 8:30 a.m.

Caterpillar, Inc., is one of the defendants in this litigation. By a timely filed *Frye* motion, it challenged the Faber/Maddox opinions as infirm because Caterpillar contended they rested on methodologies not generally accepted in the relevant scientific community. No other timely *Frye* Motions were filed.

On February 2, 2007, all parties appeared before the Court to address the Caterpillar motion. Plaintiff limited her presentation to argument that the doctors [1] challenged methodologies and opinions were not novel and thus not subject to a *Frye* challenge. At the close of Plaintiff's presentation, Plaintiff's counsel, contending Plaintiff had not realized the proceeding was to be an evidentiary hearing rather than an argument, requested time to supplement the record. Caterpillar objected. The Court, subject to objection, invited either party to proffer a record supplement on or before February 12, 2007. On February 12, Plaintiff proffered a supplemental record.

DISCUSSION

At the outset, the Court observes the relief it grants Caterpillar is narrowly drawn. Caterpillar seeks total pretrial exclusion of all Maddox opinions; the Court does not grant that prayer for relief. The exact Maddox opinion that Caterpillar challenges reads: "Based on the exposure history, all asbestos exposure substantially contributed and caused this lethal malignant pleural mesothelioma." The term "history," as used in this Opinion, is ambiguous. It may refer to the overall work-exposure history, without regard to individual exposures, or it may refer to the record of specific exposures developed in this case. Because the Plaintiff at hearing could not clarify this ambiguity, the Court's opinion is confined to a possible opinion that any asbestos exposure in a work-exposure history regardless of its nature or extent, is a competent legal cause of the disease process.

Under *Frye,* novel scientific evidence is admissible if the methodology that underlies the evidence has general acceptance in the relevant scientific community. Commonwealth v. Blasioli, 713 A. 2d. 1117, 1119 (Pa. 1998). The "general acceptance" test is limited to methodology, not conclusions. Where *Frye* is properly at issue, the proponent of the evidence bears the burden of proving that the methodology an expert used is generally accepted by scientists in the relevant field as a method for arriving at the conclusion the expert will testify to at trial. Grady v. Frito-Lay, Inc., 839 A. 2d 1038 (Pa. 2003).

Plaintiff argues no *Frye* issue exists with respect to the Maddox opinion because it is not novel. To support her position, Plaintiff points to several cases where on review our appellate courts upheld trial court introductions of opinions similar to those Dr. Maddox now proffers. See, e.g., Smalls v. Pittsburgh Corning, et. al., 843 A. 2d 410 (Pa. Super. 2004); Andalero v. Armstrong World Industries, Inc., 799 A. 2d 71 (Pa. Super. 2002); Cauthorn v. Owens Corning Fiberglas Corp., 840 A. 2d 1028 (Pa. Super. 2004).

For several reasons, the Court finds these cases inapplicable to the *Frye* determination now before it.

First, none of these cases involve *Frye* challenges. A *Frye* challenge is distinct from other expert-driven challenges, such as qualifications per Pa. R.E. 702. Grady, 839 A.2d at 1045.

Second, none of these cases involved challenges to methodologies, which is the heart of a *Frye* challenge. For *Frye* purposes, "methodology" refers to the scientific method, a method of research in which a problem is identified, relevant data are gathered, a hypothesis is formulated from these data, and the hypothesis is empirically tested. Trach v. Fellin, 81 7 A. 2d 1102 (Pa. Super. 2003).

And third, by offering these cases, Plaintiff advances a definition of "novel" which is at odds with that term as used in *Frye.* "Novel" and "new" are not synonyms in this area of the law. Astrology and lie detector tests, for example, can be novel, within the meaning of *Frye,* even though these methods have been around for many years and cannot qualify as "new." Commonwealth v. Dengler, 843 A. 2d 1241 (Pa. Super. 2004), *affirmed* 586 Pa. 54, 890 A. 2d 372 (2005).

All Plaintiff's cases show is that opinions similar to Dr. Maddox's have been around for some time. The cases cited do not support a finding that the methodology Dr. Maddox used in this case is generally accepted in the relevant scientific community. Although Trach involves the dose-response of linking toxic exposure to harmful effects, this case does not address the downward extrapolation methodology Dr. Maddox appears to offer here. The Court thus finds the methodologies advanced in the case *sub judice* are novel within the meaning of *Frye.*

The Court now considers what happened, and did not happen, at the *Frye* hearing in this case.

Plaintiff presented no evidence on the issue of general acceptance. Normally, because Plaintiff has the burden of proof on this matter, a ruling in favor of Caterpillar would automatically follow. Plaintiff asked the Court to accept her supplemental record because counsel said they were confused about the purpose of the February 2 court proceeding.

The Court does not understand counsel's confusion. August 15, 2006, Order unambiguously refers to the February 2 *Frye* proceeding as a hearing. Plaintiff and defense counsel understood this date was preset to allow all parties to schedule attendance for any pretrial *Frye* dispositions.

Nonetheless, rather than grant Caterpillar's motion out of hand because of the inadequate record, the Court over objection permitted Plaintiff to proffer a post-hearing submission. In response to that invitation, Plaintiff now offers hundreds of pages of text, including deposition transcripts, affidavits, reports, and test results from various sources.

At the outset, the Court disapproves of Plaintiff's approach to making a *Frye* record. She easily could have prepared these submissions for the February 2 hearing. Had Plaintiff done so, Caterpillar then would have had an opportunity to raise objections, particularly with respect to deposition transcripts. Instead, the Court is left to guess what portions of Plaintiff's proffer would be proper for it to consider.

In this situation, the Court easily could sustain Caterpillar's objection and reject the entire proffered record. But that is not necessary here, because the Court finds the record offered does not support a finding of general acceptance.

Dr. Maddox's challenged opinion, and its supporting assumptions, can be summarized as follows: 1) there is no “safe” minimum level of asbestos exposure, 2) asbestos exposure is a recognized cause of mesothelioma, and 3) the etiology of mesothelioma results from the cumulative process of asbestos exposure (or, the greater the overall exposure, the greater the probability mesothelioma will develop in a particular case); therefore 4) any exposure to a product shedding asbestos fibers must be deemed a legal cause of an individual's mesothelioma.

This chain of ideas is not methodology at all, but an effort at logical analysis. As logic, the effort fails. The three premises Dr. Maddox advances - asbestos is an unsafe product, asbestos exposure is a recognized cause of mesothelioma, and the greater the asbestos exposure, the greater the risk of contracting mesothelioma - do not support a leap to the conclusion that any asbestos exposure must be deemed a legal cause of the resulting harm, especially as Pennsylvania defines the term “legal cause.”

Under Pennsylvania law, legal cause, also known as substantial contributing factor, requires proof of direct causation. In the context of this case, Plaintiff must prove decedent's exposure to Caterpillar's asbestos-shedding processes was a direct cause of his mesothelioma. If exposure to Caterpillar products, along with other legally-sufficient exposures, contributed to the single, indivisible harm of mesothelioma, then all causal actors shall be treated as joint and several tortfeasors. *Martin v. Owens-Corning Fiberglass,* 528 A.2d 947 (Pa. 1987); § 433 A, Restatement (Second) of Torts.

The need for proof of direct process is underscored by the leading case of *Eckenrod v. GAF Corp*. 544 A.2d 50 (Pa.Super. 1998). In this case, which sets a widely used summary judgment standard in asbestos litigation, our Superior Court recognized that it is virtually impossible in asbestos litigation to establish a sole causal link between the victim's exposure to asbestos fibers shed by a particular defendant's product and a resultant disease process. Therefore, that court held: “Whether a plaintiff could successfully get to a jury or defeat a motion for summary judgment by showing circumstantial evidence depends upon the frequency of the use of the product and the regularity of Plaintiff's employment in proximity thereto.” *Eckenrod,* 544 A.2d at 53.

The Eckenrod frequency/proximity standard does not negate the requirement of direct causation; rather, it permits a finding of direct causation by inference. The Maddox opinion, to the extent it offers up a legal conclusion, does not comport with *Eckenrod* or *Martin.* Further, to the extent it offers a medical conclusion, it inescapably advances the “single fiber” theory, or a very close variant.

The “single fiber” theory holds that exposure to a single asbestos fiber can cause mesothelioma and other disease processes. Calling it a “cumulative exposure” theory does not alter its operative effect. For example, in the context of this case, Dr. Maddox opines that, because asbestos exposure is inherently dangerous, asbestos exposure causes mesothelioma, and the disease process is cumulative (that is, the greater the exposures, the greater the risk) if Plaintiff's decedent had been exposed to twenty different

asbestos-shedding products, and Caterpillar were one of those products, the Caterpillar exposure would be a legal cause of the Plaintiff's decedent's mesothelioma, even if that exposure consisted of a single asbestos fiber.

As the Court previously has noted, Dr. Maddox's non-scientific assumptions do not logically lead to a single-fiber hyposis, nor does the theory square up with *Eckenrod's* legal proof requirements. So the question remains: is the "single-fiber" opinion supported by a methodologically that is generally accepted in the relevant scientific community?

The record in this case offers no methodology to support a "single fiber" opinion, much less general acceptance of any such methodology. In particular, Plaintiff's record does not meet her burden of proving that either extrapolation-downward dose-response or case studies are generally accepted methodologies in the relevant scientific community to project harm from "extremely low" or "low" doses of toxic exposure.

The Court having found Plaintiff's methodology is novel, and Plaintiff having failed to meet her burden of general acceptance,

The Court's Order follows.

ORDER OF COURT

AND NOW, February 22, 2007, it is ORDERED, that Defendant's *Frye* Motion to Exclude Testimony of Dr. John C. Maddox is granted in part. By this Order, Dr. Maddox is precluded from offering an opinion that, regardless of specific exposure history, all asbestos exposures substantially contributed and caused the Plaintiff's Decedent's mesothelioma.

BY THE COURT;

_____

Gregory A. Olson, Judge

Footnotes

1 Plaintiff has since withdrawn Dr. Faber from her witness list)

EXHIBIT 6

2013 WL 2477077
Only the Westlaw citation is currently available.
United States District Court,
C.D. California.

David SCLAFANI, et al.
v.
AIR AND LIQUID SYSTEMS CORP., et al.

Nos. 2:12–cv–3013–SVW–PJW, 2:12–cv–3037–SVW–PJW. | May 9, 2013.

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Opinion

Proceedings: IN CHAMBERS ORDER Re DEFENDANTS' MOTIONS IN LIMINE Case No. 2:12–cv–3013–SVW–PJW: [170][172][179][180][181][182][183][184][221] Case No. 2:12–cv–3037–SVW–PJW: [116][117][118][119][120][121][122][132][133]

STEPHEN V. WILSON, District Judge.

***1** Paul M. Cruz, Deputy Clerk.

Defendants' Motion in Limine Number 1 **(Dckt.179)** [1]

Defendants' Motion in Limine Number 1 seeks to limit the admission of "any evidence regarding past medical expenses of David Sclafani through documents, expert testimony, or otherwise, to only those amounts *actually paid* by or on" Sclafani's behalf. Most of Sclafani's medical costs have been covered by the Veteran's Administration. Under California law, a tortuously injured plaintiff whose medical bills are paid by another—such as the plaintiff's health insurer—cannot recover damages for those *past* medical expenses "for the simple reason that the injured plaintiff did not suffer any economic loss in that amount." *Howell v. Hamilton Meats & Provisions, Inc.,* 52 Cal.4th 541, 548, 129 Cal.Rptr.3d 325, 257 P.3d 1130 (2011). Therefore, Sclafani will only be permitted to recover past medical expenses he *actually* paid; any other medical expenses cannot be recovered from Defendants. Plaintiffs do not oppose this portion of Defendants' Motion in Limine Number 1; therefore, to the extent they seek reimbursement for Sclafani's past medical expenses, Plaintiffs will be limited to introducing evidence of amounts actually paid by Sclafani.

Defendants' Motion in Limine Number 1 also seeks to exclude the opinions of Plaintiffs' economist Dr. David Fractor as speculative an unfounded. At trial, Fractor will opine that Plaintiff would lose future earnings of $68,463 and lost "household services" of $180,233.

"Where lost future earnings are at issue, an expert's testimony should be excluded as speculative if it is based on unrealistic assumptions regarding the plaintiff's future employment prospects." *Boucher v. U.S. Suzuki Motor Corp.,* 73 F.3d 18, 21 (2d Cir.1996). Here, Dr. Fractor's $68,463 figure was calculated by *assuming* that Sclafani would work to the end of his life and earn the New Hampshire minimum wage of $7.25 an hour. However, prior to his mesothelioma diagnosis, Sclafani was selfemployed, and his business was generating no income. Moreover, Plaintiffs have provided no indication that Sclafani—who is at least 70 years old—intended on taking up a full-time, minimum wage job. Fractor's calculations are, by definition, speculative—they are not based either on Sclafani's work history, nor his stated intentions to return to work. Thus, Dr. Fractor will not be permitted to testify as to Sclafani's lost future earnings.

However, Dr. Fractor will be permitted to testify as to the lost "household services." Defendants argue that Dr. Fractor failed to account for the fact that, since Sclafani has become sick, his wife has taken over the household responsibilities from Sclafani. This argument was specifically rejected by the California Court of Appeals in *McKinney v. California Portland Cement Co.,* 96 Cal.App.4th 1214, 1228, 117 Cal.Rptr.2d 849 (2002). Defendants present no other objection to Dr. Fractor's methodology as to Plaintiffs' "household services" claim; therefore, Dr. Fractor will be permitted to testify on this point at trial.

***2** Thus, Defendants' Motion in Limine Number 1 is GRANTED IN PART, AND DENIED IN PART.

Defendants' Motion in Limine Number 2 **(Dckt.179–1)**
Defendants' Motion in Limine Number 2 is a motion by defendant Foster Wheeler that seeks to exclude any testimony "purporting to identify a Foster Wheel product solely by testimony that its name appeared [on a product], and any testimony relying on such identification" as either inadmissible hearsay or on the basis of the best evidence rule. [2] As this Court determined in ruling on Buffalo's motion for summary judgment, the Ninth Circuit has held that labels affixed to a medium "are most appropriately characterized as circumstantial evidence of origin, rather than as an 'assertion' within the meaning of the hearsay rule." *Los Angeles News Serv. v. CBS Broad., Inc.,* 305 F.3d 924, 935 *opinion amended and superseded,* 313 F.3d 1093 (9th Cir.2002) (finding that an identifying "CBS" slate appearing on the opening frames of a videotape is not hearsay and 'is more akin to a postmark or timestamp" such that it is an "indicia of origin" that did not implicate the hearsay rule); *see also United States v. Snow,* 517 F.2d 441, 443 (9th Cir.1975) (holding that a piece of tape affixed to a briefcase with the name "Bill Snow" printed on it was *not* hearsay, but rather circumstantial evidence that the briefcase belonged to Bill Snow).

However, as this Court observed in its separate order of May 9, 2013, Sclafani's testimony that he saw the words "Foster Wheeler" is subject to the best evidence rule. The Court will defer ruling on Foster Wheeler's motion to exclude this portion of Sclafani's testimony on the basis of the best evidence rule until the pretrial conference set for May 13, 2013.

Defendants' Motion in Limine Number 3 **(Dckt.179–2)**
Defendants' Motion in Limine Number 3 is a motion by defendant Foster Wheeler that seeks to exclude the testimony of Plaintiffs' expert Captain Francis Burger altogether. Foster Wheeler argues that Captain Burger's expert report fails to indicate that he reviewed "any materials regarding Foster Wheeler in preparation for this case." However, in his expert report, Captain Burger states that he reviewed, among other things, the discovery responses of "Naval Defendants"—including Foster Wheeler—and deposition transcripts of the Naval Defendants' "Person Most Qualified," and the ship records for the Naval vessels that Sclafani worked on, which noted, among other things, that there were Foster Wheeler boilers aboard the USS Morton. *See* Burger Rept. at 11–12.

In the alternative, Foster Wheeler asks this Court to limit Captain Burger's testimony to the opinions disclosed in his expert report, and to exclude any opinions that lack a factual basis. Foster Wheeler argues that many the opinions Captain Burger offered in his declaration in support of Plaintiffs' motion for summary judgment were either not disclosed in Captain Burger's expert report or lack a factual basis. Specifically, Foster Wheeler attacks Captain Burger's opinions 1) that Foster Wheeler boilers were often supplied with asbestos-containing parts already installed; 2) that Foster Wheeler supplied spare parts, including gaskets, for use in and with its boilers; 3) that during Sclafani's service aboard the USS Morton, Foster Wheeler's boilers onboard the Morton "would more likely than not have been insulated with asbestos containing insulation and utilized asbestos containing refractory material, gaskets, and packing;" and 4) that it was more likely than not that Sclafani would have removed and replaced asbestos-containing insulation, refractory material, gaskets, or packing supplied by Foster Wheeler.

***3** Plaintiffs have failed to file an opposition to Defendants' Motion in Limine Number 3. Without greater guidance from Plaintiffs, the Court cannot discern whether Captain Burger included each of these opinions in his initial expert report, nor whether his opinions were formed the factual bases for each opinion. Thus, Plaintiffs shall have until Monday, May 13, 2013 at 10:00 a.m. to respond to Defendants' Motion in Limine Number 3, and to identify where in Captain Burger's report he disclosed these four opinions, and his factual basis for so opining.

Defendants' Motion in Limine Number 4 **(Dckt.179–3)**
Defendants' Motion in Limine Number 4 seeks to preclude Plaintiffs from introducing "all evidence post-dating

Sclafani's last alleged exposure to asbestos" as irrelevant and as barred by Federal Rule of Evidence 407. Defendants have failed to identify which items of evidence they are seeking to exclude, and thus will defer ruling on this motion until specific items of evidence are identified. However, the Court notes that, to the extent that Plaintiffs seek to introduce evidence of remedial measures taken by Defendants after Sclafani's alleged exposure to asbestos (i.e., post–1963), such evidence will be excluded under Rule 407. *See also* Pls.' Mot. in Limine No. 3.

Defendants' Motion in Limine Number 5 **(Dckt.179–4)**
Defendants' Motion in Limine Number 5 seeks to preclude Plaintiffs' expert Dr. Barry Horn from testifying regarding the cost or value of Sclafani's medical treatment. Defendants' argue that 1) any such opinion was not included in Dr. Horn's Rule 26 report; and 2) if he attempts to amend his previous report, he has failed to review Sclafani's medical records. Plaintiffs concede that Dr. Horn has *not* reviewed Sclafani's medical records; therefore, Dr. Horn has no factual basis from which he could opine as to the cost or value of Sclafani's medical treatment.

Therefore, Defendants' Motion in Limine Number 5 is GRANTED.

Defendants' Motion in Limine Number 7 **(Dckt.179–5)** [3]
Defendants' Motion in Limine Number 7 is a motion by defendant Foster Wheeler that seeks to preclude Plaintiffs from making any argument that Foster Wheeler is liable for asbestos-containing packing and gaskets that it did not supply or distribute. As this Court previously found, Plaintiffs' claims against Foster Wheeler is premised on their argument that Sclafani was exposed either to the original asbestos-containing parts in the Foster Wheeler boilers, or Foster Wheeler-supplied spares during Sclafani's service on the Morton. Plaintiffs' argument at trial will be limited to these theories of liability. [4]

Defendants' Motion in Limine Number 8 **(Dckt.179–6)**
Defendants' Motion in Limine Number 8 seeks to bifurcate the liability and damages phases of the trial. The Court will phase the trial in the matter discussed with the parties; thus, the motion is DENIED as MOOT.

Defendants' Motion in Limine Number 9 **(Dckt.116)**

***4** Defendants' Motion in Limine Number 9 seeks to preclude Plaintiffs from offering expert testimony and documents regarding the "historical development of medical and scientific information the purported dangers of asbestos exposure." Defendants do not identify which specific items of evidence they seek to exclude; as such, the Court will defer ruling on this motion until trial.

Defendants' Motion in Limine Number 10 **(Dckt.132)**
Defendants' Motion in Limine Number 10 seeks to preclude Plaintiffs from eliciting opinions from their experts that "every" exposure to asbestos is a substantial factor in causing mesothelioma. Specifically, Defendants object to the opinion of Dr. Arnold Brody, who intends to opine that "[o]nce a person develops an asbestos-related cancer, it is not possible to exclude any of the person's abovebackground exposures to asbestos from the causal chain. Each and every exposure to asbestos that an individual with mesothelioma experienced in excess of a background level contributes to the development of the disease."

The question of causation in asbestos-related litigation is an exceedingly difficult one. "At the most fundamental level, there is scientific uncertainty regarding the biological mechanisms by which inhalation of certain microscopic fibers of asbestos leads to lung cancer and mesothelioma." *Rutherford v. Owens–Illinois, Inc.,* 16 Cal.4th 953, 974, 67 Cal.Rptr.2d 16, 941 P.2d 1203 (1997). The California Supreme Court addressed this difficulty by articulating a two-part causation test: first, the plaintiff must "establish some threshold *exposure* to the defendant's defective asbestos-containing products [.]" *Rutherford,* 16 Cal.4th at 982, 67 Cal.Rptr.2d 16, 941 P.2d 1203 (footnote omitted). Second, a plaintiff must establish to a "reasonable medical probability that a particular exposure or series of exposures was a 'legal cause' of his injury, i.e., a *substantial factor* in bringing about the injury." *Id.* This latter inquiry requires a plaintiff to show that his or her exposure to a particular defendant's asbestos-containing product, "in reasonable medical probability," was a substantial factor in contributing to the "aggregate *dose* of asbestos the plaintiff or decedent inhaled or ingested." *Id.* at 976–77, 67 Cal.Rptr.2d 16, 941 P.2d 1203.

Dr. Brody's opinion—that "each and every exposure ... contributes to the development of" mesothelioma—is, in fact, the legal conclusion that, under *Rutherford,* a jury must reach. While an opinion is "not objectionable just because it embraces an ultimate issue," *see* Fed.R.Evid. 704, the Court

finds that this opinion should be excluded for two other reasons.

First, as a legal issue, accepting Dr. Brody's opinion as true would render the "substantial factor" prong of the causation test meaningless. If "each and every exposure" is a *substantial factor* in leading to the development of mesothelioma, then all a plaintiff would have to do is prove 1) that he had mesothelioma; and 2) that he was exposed to asbestos at some time. Similar opinions have been rejected on precisely this basis. *Lindstrom v. A–C Prod. Liab. Trust,* 424 F.3d 488, 492–3 (6th Cir.2005) (upholding the district court's exclusion of an "each and every exposure" opinion and holding that " '[m]inimal exposure' to a defendant's product is insufficient[,]" as"[a] holding to the contrary would permit imposition of liability on the manufacturer of any product with which a worker had the briefest of encounters on a single occasion."); *see also Holcomb v. Georgia Pac., LLC,* 289 P.3d 188, 197 (Nev.2012) (Noting that courts that adopt "the three-factor test of frequency, regularity, and proximity" in determining "substantial factor" regularly "reject the 'any' exposure argument.").

***5** Secondly, Plaintiffs have failed to carry their burden of demonstrating this opinion is relevant and reliable, as required by Federal Rule of Evidence 702 and *Daubert v. Merrell Dow Pharmaceuticals,* 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993). "Under *Daubert,* the trial court must act as a 'gatekeeper' to exclude junk science that does not meet Federal Rule of Evidence 702's reliability standards by making a preliminary determination that the expert's testimony is reliable." *Ellis v. Costco Wholesale Corp.,* 657 F.3d 970, 982 (9th Cir.2011). In making this determination, district courts are to consider, among other things, "(1) whether the scientific theory or technique can be (and has been) tested, (2) whether the theory or technique has been subjected to peer review and publication, (3) whether there is a known or potential error rate, and (4) whether the theory or technique is generally accepted in the relevant scientific community." *Elsayed Mukhtar v. California State Univ., Hayward,* 299 F.3d 1053, 1064 (9th Cir.2002) *amended sub nom. Mukhtar v. California State Univ., Hayward,* 319 F.3d 1073 (9th Cir.2003) (citing *Daubert,* 509 U.S. at 593–94.).

Plaintiffs have failed to demonstrate that Dr. Brody's opinion is the product of reliable techniques. It is unclear how Dr. Brody came to his "every exposure" opinion; although he refers to several studies (none of which was provided to the Court by Plaintiffs), each study concludes only that "no amount of exposure to asbestos above the background levels present in ambient air has been established as too low to induce mesothelioma." Most troubling is Dr. Brody's own testimony—when cross-examined in another action about his "each and every exposure" opinion, Dr. Brody conceded that 1) there was no data to establish that all exposures contribute to mesothelioma; 2) his theory could not be tested; 3) his theory had not been published in any peer-review literature; and 4) had not been "put together as a scientific principle and tested." *See* Decl. of Crane's Counsel Bradley W. Gunning ¶ 8, Ex. G. These admissions demonstrate that, in forming his theory, Dr. Brody has not, and indeed cannot, met at least two of the four criteria *Daubert* sets forth in assessing a theory's reliability. Thus, the Court GRANTS Defendants' Motion in Limine Number 10.

Defendants' Motion in Limine Number 11 **(Dckt.121)**

Defendants' Motion in Limine Number 11 seeks to preclude Plaintiffs from introducing evidence or making argument that any Defendant manufactured or supplied asbestos-containing products not at issue in this case—for example, Defendants' marketing materials, product catalogues, patents, technical drawings and purchasing specifications regarding products *not* identified by Sclafani or witnesses as the source of Sclafani's asbestos exposure. Plaintiffs argue that this evidence is relevant to establish that Defendants should have known of the dangers of asbestos, an element of their negligence and strict liability claims.

***6** Defendants have failed to identify which items of evidence they are seeking to exclude; thus, the Court will defer ruling on this motion until Plaintiffs seek to introduce specific items of evidence.

Defendants' Motion in Limine Number 12 **(Dckt.133)**

Defendants' Motion in Limine Number 12 relates to studies conducted by Material Analytical Services ("MAS"). The MAS studies purported to measure the amount of airborne asbestos fibers created by the removal and wire-scraping of packing and gaskets from valves. Defendants claim that the techniques and methodologies are at odds with the generally accepted scientific methods for making such measurements, and should therefore be excluded under *Daubert v. Merrell Dow Pharmaceuticals,* 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993) and its progeny. Defendants present substantial evidence that the techniques used in the MAS study are at odds with the Occupational Safety and Health Administration's methods for measuring

exposure, and therefore are *not* the product of "reliable principles and methods," a prerequisite of the introduction of expert testimony under Rule 702. Moreover, several courts have excluded this study on these grounds. Plaintiffs' have submitted no opposition to the motion; [5] therefore, Defendants' Motion in Limine Number 12 is GRANTED.

Defendants' Motion in Limine Number 13 **(Dckt.117)**

Defendants' Motion in Limine Number 13 seeks to preclude Plaintiffs from introducing evidence of a test performed at a Shell Oil Company plant in which a "Durabla" [6] gasket was removed with a power grinder (Defendants do not indicate what the gasket was removed from). Plaintiffs intend to use this study under to cross-examine Defendants' expert witnesses' opinions that the removal of gaskets "emits practically no asbestos dust." Defendants appear to object to the introduction of this study on hearsay grounds: although statements contained in a "treatise, periodical, or pamphlet" may be introduced to cross-examine an expert witness under the learned treatise exception to the hearsay rule, *see* Fed.R.Evid. 803(18), Plaintiffs must establish that the treatise is "a reliable authority." They have failed to do so here: the study does not describe the methodology used; it was not published in a journal; and the person who performed the test is not available for cross-examination. Thus, Defendants' Motion in Limine Number 13 is GRANTED.

Defendants' Motion in Limine Number 14 **(Dckt.118)**

Defendants Motion in Limine Number 14 seeks to exclude "any evidence, reference, or argument relating to the Southern Power and Industry Trade Journal." Plaintiffs have agreed to the granting of this motion; therefore, Defendants' Motion in Limine Number 14 is GRANTED.

Defendants' Motion in Limine Number 15 **(Dckt.122)**

Defendants' Motion in Limine Number 15 seeks to preclude Plaintiffs from introducing documents "relating to trade organizations of which Defendant were never members," such as the National Safety Council. Plaintiffs intend on introducing the identified documents as learned treatises to cross-examine Defendants' experts, specifically as to the issue of what was *knowable* about the dangers of asbestos. Such evidence is plainly relevant to an element of (at least) Plaintiffs' strict liability and negligence failure to warn claims. *See* CACI 1222 (listing the elements of negligent failure to warn, including "that Defendant knew or *reasonably should have known that the product was dangerous* or was likely to be dangerous when used or misused in a reasonably foreseeable manner") (emphasis added); *see also* CACI 1205 (listing the elements of strict liability failure to warn, including that the product "had potential risks that were known or *knowable in light of the generally recognized and prevailing best scientific and medical knowledge available at the time of manufacture, distribution, or sale"*) (emphasis added).

***7** Defendants' further argue that Plaintiffs cannot authenticate these documents; however, these reports can likely be authenticated either by the testimony of a witness with knowledge (namely, Defendants' experts), *see* Fed.R.Evid. 901(b)(1), or as a self-authenticating newspaper or periodical. *See* Fed.R.Evid. 902(6). Thus, Defendants' Motion in Limine Number 15 is DENIED.

Defendants' Motion in Limine Number 16 **(Dckt.120)**

Defendants Motion in Limine Number 16 seeks to exclude "evidence or argument regarding their alleged liability for other manufacturer's products." More specifically, Defendants seek to exclude evidence of exposure to asbestos-products that were not designed, manufactured, supplied or otherwise placed into the stream of commerce by Defendants. [7] Plaintiffs have agreed to the granting of this motion; therefore, Defendants' Motion in Limine Number 16 is GRANTED.

Defendants' Motion in Limine Number 17 **(Dckt.119)**

Defendants' Motion in Limine Number 17 seeks to preclude Plaintiffs' expert Captain Francis Burger from opining that equipment manufacturers (such as Defendants) were "required by Navy specifications to warn of the dangers of death and personal injury from asbestos released from the foreseeable work practices involved in installing, repairing, and removing such equipment," because this opinion lacks foundation. This motion also seeks to preclude Captain Burger from opinion that the Navy selected replacement gaskets based on information in the equipment manufacturers' "drawings and technical manuals" and that the Navy "utilized the original equipment manufacturers for replacement parts."

The Court remains unclear how these opinions are relevant to the instant action, and will thus defer ruling on this motion in limine until the May 13, 2013 pretrial conference.

Defendants' Motion in Limine Number 18 **(Dckt.180)**

Defendants' Motion in Limine Number 18 is a motion by defendant Buffalo Pumps that seeks to preclude Plaintiffs' expert Captain Francis Burger from offering opinions at trial that were not included in his Rule 26 report. Specifically, Buffalo Pumps argues that Captain Burger's Rule 26 report did not include an opinion about "spare" gaskets, only "replacement" gaskets.

As this Court previously found, Buffalo Pumps did not manufacture the asbestos-containing packing and gaskets at issue in this action; instead, Buffalo Pumps' potential liability was premised on Plaintiffs' theory that Buffalo Pumps supplied these products. There were three potential avenues through which Buffalo Pumps may have supplied the packing and gaskets: either encased in the original pumps aboard the Rogers or the Morton; as "spares" that Buffalo Pumps supplied with the originally installed parts; and as "replacement" parts. This Court found that, as a matter of law, Sclafani could not have been exposed to asbestos from the original packing or gaskets, and that Buffalo Pumps did not supply "replacements," but that a triable issue remained as to whether Sclafani worked with Buffalo Pumps-supplied *spares*.

***8** Integral to the Court's finding was Captain Burger's opinion that "Buffalo provided asbestos-containing spare packing and gaskets for originals [,]" and that the Navy would "use manufacturer-supplied spare parts as replacements for original parts prior to dipping into the general supply stock." *See* Burger Decl. in Opp. to Buffalo Pumps' MSJ ¶ 21. Buffalo Pumps now contends that this opinion was not previously disclosed in Captain Burger's Rule 26 report, and is based on Captain Burger's review of materials not previously disclosed, and that the opinion should be excluded as prejudicial. [8]

In his Rule 26 report, Captain Burger discusses "replacement" parts, but does not discuss "spares" provided by Buffalo Pumps. *See* Capt. Burger's Expert Report at pp. 18, 19 (noting that "the industry utilized the original equipment manufacturers [such as Buffalo Pumps] for *replacement* parts, including asbestos gaskets and packing"). Plaintiffs argue that the word "replacement" is the same as "spare," and thus Burger's opinion was adequately disclosed.

The Court has serious reservations about Plaintiffs' argument. [9] However, it appears that this failure was not prejudicial; during Captain Burger's deposition, Buffalo Pumps' counsel appeared to distinguish between "spare gaskets" and "subsequent replacement parts." It appears from this portion of the deposition that Buffalo Pumps was aware that Captain Burger intended to opine as to the provision of "spares," and thus the alleged failure to disclose was not prejudicial. Therefore, Defendants' Motion in Limine Number 18 is DENIED.

Defendants' Motion in Limine Number 19 **(Dckt.181)**

Defendants' Motion in Limine Number 19 is a motion by defendant Buffalo that seeks to preclude Sclafani from testifying that he saw the name "Buffalo Pumps" or "Buffalo" on the materials he worked with during his time aboard the USS Morton, as either inadmissible hearsay or subject to the best evidence rule. As this Court determined in ruling on Buffalo's motion for summary judgment, the Ninth Circuit has held that labels affixed to a medium "are most appropriately characterized as circumstantial evidence of origin, rather than as an 'assertion' within the meaning of the hearsay rule ." *Los Angeles News Serv. v. CBS Broad., Inc.,* 305 F.3d 924, 935 *opinion amended and superseded,* 313 F.3d 1093 (9th Cir.2002) (finding that an identifying "CBS" slate appearing on the opening frames of a videotape is not hearsay and 'is more akin to a postmark or timestamp" such that it is an "indicia of origin" that did not implicate the hearsay rule); *see also United States v. Snow,* 517 F.2d 441, 443 (9th Cir.1975) (holding that a piece of tape affixed to a briefcase with the name "Bill Snow" printed on it was *not* hearsay, but rather circumstantial evidence that the briefcase belonged to Bill Snow).

However, as this Court observed in its separate order of May 9, 2013, the words "Buffalo" and "Buffalo Pumps" are subject to the best evidence rule. The Court will defer ruling on Buffalo's motion on this basis until the pretrial conference set for May 13, 2013.

***9** Buffalo also seeks to preclude Sclafani from opining that Buffalo Pumps was the "source or origin" of the "spare" parts Sclafani worked with. Buffalo is correct that Sclafani is unqualified to give such an opinion: he was not involved in the Naval supply chain, and has no knowledge of where the packing and gaskets he worked with came from. Thus, Sclafani will not be permitted to opine that the packing and gaskets he worked with were supplied by Buffalo.

Thus, Defendants' Motion in Limine Number 19 GRANTED in part.

Defendants' Motion in Limine Number 20 **(Dckt.184)**
Defendants' Motion in Limine Number 20 is a motion by Defendant Buffalo Pumps that seeks to preclude the introduction of any evidence or argument of Sclafani's exposure to any asbestos-containing gaskets or packing allegedly supplied by Buffalo aboard the USS Rogers because that issue was "finally and fully adjudicated" when this Court granted in part Buffalo Pumps' motion for summary judgment. [10] Plaintiff does not oppose the motion; therefore, Defendants' Motion in Limine Number 20 is GRANTED.

Defendants' Motion in Limine Number 21 **(Dckt.183)**
Defendants' Motion in Limine Number 21 is a motion by defendant Buffalo Pumps that seeks to preclude the introduction of any evidence or argument of Sclafani's exposure to any asbestos-containing gaskets or packing during his time aboard the USS Morton that were either originally-installed in the Buffalo Pumps-supplied pumps, or were supplied as "replacement" parts to those pumps, because these issues were "finally and fully adjudicated" when this Court granted in part Buffalo Pumps' motion for summary judgment. [11] Plaintiff does not oppose the motion; therefore, Defendants' Motion in Limine Number 21 is GRANTED.

Defendants' Motion in Limine Number 22 **(Dckt.182)**
Defendants' Motion in Limine Number 22 is a motion by Defendant Buffalo Pumps that seeks to preclude the introduction of a declaration signed by Sclafani that was submitted in opposition to Buffalo Pumps' motion for summary judgment as hearsay, pursuant to Federal Rules of Evidence 801 and 802. Plaintiff does not oppose the motion; therefore, Defendants' Motion in Limine Number 22 is GRANTED.

Defendants' Motion in Limine Number 23 **(Dckt.170)**
Defendants' Motion in Limine Number 23 is a motion by defendant Goodyear that seeks to preclude Plaintiffs from introducing evidence or making argument that Goodyear manufactured asbestos-containing products *other* than the ones to which Sclafani alleges he was exposed. This motion is the same as Defendants' Motion in Limine number 11, which sought to exclude evidence all evidence of *any* defendant "manufactured or supplied asbestos-containing products not at issue in this case." Here, as there, Goodyear has failed to identify which items of evidence it seeks to exclude; thus, the Court will reserve ruling on this motion until Plaintiffs seek to introduce specific items of evidence.

Defendants' Motion in Limine Number 24 **(Dckt.172)**
***10** Defendants' Motion in Limine Number 24 is a motion by defendant Goodyear that seeks to preclude Plaintiffs, their counsel, and their expert witnesses from "making any reference to asbestos exposure from new Goodyear gasket material after 1969," and from making any reference to "any Goodyear-related documents, manuals, or any other written materials relating to any gasket product manufactured by Goodyear after 1969." Plaintiffs do not oppose the motion; therefore, Defendants' Motion in Limine Number 24 is GRANTED.

Defendants' Motion in Limine Number 25 **(Dckt.221)**
Defendants' Motion in Limine Number 25 is a motion to preclude Plaintiffs from using any graphic and illustrative material not timely disclosed. Defendants do not identify what, if any material, they are seeking to exclude; thus, the Court defers ruling on this motion until such material is identified. If Plaintiffs attempt to use graphs, pictures, or other illustrations at trial that were not disclosed at least eleven (11) days before trial (or before May 3, 2013), this Court will prohibit Plaintiffs from using these materials under Local Rule 16–3. ("If not already disclosed ... the parties shall disclose copies of all graphic or illustrative material to be shown the trier of facts as illustrating the testimony of a witness at least eleven (11) days before trial.").

Footnotes

1 All docket numbers refer to case number 2:12–cv–3013–SVW–PJW, except for the motions in limine numbers 9 through 17, which were filed under case number 2:12–cv–3037–SVW–PJW.

2 Foster Wheeler also seeks to strike portions of Sclafani's deposition that were elicited "through impermissible leading questions." At the pretrial conference, Plaintiffs indicated that Sclafani would not be testifying himself; instead, his testimony will be offered by reading his deposition into the record. Plaintiffs' counsel is currently identifying which portions of Sclafani's deposition they seek to introduce; and Foster Wheeler (among others) will respond by identifying, and objecting to, specific items of testimony. The Court will defer on ruling on this objection until this process is complete.

Foster Wheeler also argues that Sclafani's identification of Foster Wheeler gaskets and packing during his deposition lacked foundation and was based on speculation. Specifically, they point out that, while at certain points in his deposition Sclafani recalled seeing the name "Foster Wheeler" on boilers, gaskets, and packing, at other points he could not remember seeing any such logos or writing. These arguments obviously go to Sclafani's credibility, an issue reserved for the jury.

3 Defendants did not file a Motion in Limine Number 6.

4 Foster Wheeler's motion was likely made to preempt Plaintiffs from arguing that Foster Wheeler is liable because it "was foreseeable [to Foster Wheeler that] workers would be exposed to and harmed by the asbestos in replacement parts and products used in conjunction with their pumps and valves." *O'Neil v. Crane Co.,* 53 Cal.4th 335, 342, 135 Cal.Rptr.3d 288, 266 P.3d 987 (2012). However, in *Crane,* the California Supreme Court explicitly rejected this theory of liability, holding that a plaintiff in an asbestos-related personal injury suit must show that the defendant being sued was somehow involved in the manufacturing, distribution, or retail chain of the asbestos product to which a plaintiff was exposed. *Id.* at 362–63, 135 Cal.Rptr.3d 288, 266 P.3d 987.

5 Under Local Rule 7–12, the failure to file any required document "may be deemed consent to the granting or denial of the motion."

6 Durabla was another gasket manufacturer who included asbestos in their gaskets.

7 Defendants' motion was likely made to preempt Plaintiffs from arguing that Foster Wheeler is liable because it "was foreseeable [to Defendants that] workers would be exposed to and harmed by the asbestos in replacement parts and products used in conjunction with their pumps and valves." *O'Neil v. Crane Co.,* 53 Cal.4th 335, 342, 135 Cal.Rptr.3d 288, 266 P.3d 987 (2012). However, in *Crane,* the California Supreme Court explicitly rejected this theory of liability, holding that a plaintiff in an asbestos-related personal injury suit must show that the defendant being sued was somehow involved in the manufacturing, distribution, or retail chain of the asbestos product to which a plaintiff was exposed. *Id.* at 362–63, 135 Cal.Rptr.3d 288, 266 P.3d 987.

8 Buffalo Pumps raised other objections to this portion of Captain Burger's declaration in their motion for summary judgment, objections which this Court overruled.

9 As this Court previously found, "spares" were provided by pump manufacturers, like Buffalo Pumps, when they sold the original pumps. "Replacements" were additional gaskets and packing purchased by the Navy separately-and it was undisputed that Buffalo Pumps never sold separate "replacements."

10 As this Court previously found, Plaintiffs had identified three potential sources of asbestoscontaining gaskets and packing that Buffalo Pumps might have supplied for use aboard the USS Morton during Sclafani's service aboard the ship: 1) the originally installed gaskets and packing; 2) the "replacement" gaskets and packing; and 3) the "spare" gaskets and packing. This Court concluded that no triable issue remained as to the first two sources of asbestos-containing gaskets; but that one remained as to whether Sclafani was exposed to "spare" gaskets and packing distributed by Buffalo Pumps.

11 As this Court previously found, Sclafani was *not* exposed to Buffalo-supplied asbestos-containing parts aboard the Rogers, as Sclafani did not board the Rogers until approximately eighteen years after it was commissioned.

End of Document

EXHIBIT 7

2013 WL 3179497
Only the Westlaw citation is currently available.
United States District Court,
D. Utah,
Central Division.

Arva ANDERSON, Plaintiff,
v.
FORD MOTOR COMPANY, et al., Defendants.

No. 2:06–CV–741 TS. | June 24, 2013.

Synopsis

Background: Plaintiff filed state court action alleging that his mesothelioma was caused by his exposure to asbestos. After removal, transfer by Panel on Multidistrict Litigation, and substitution of executor of plaintiff's estate, defendants moved to exclude proposed specific causation testimony.

[Holding:] The District Court, Ted Stewart, J., held that proposed testimony was not sufficiently reliable to warrant its admission.

Motion granted.

Attorneys and Law Firms

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Opinion

MEMORANDUM DECISION AND ORDER GRANTING DEFENDANTS' MOTION TO EXCLUDE THE PROPOSED SPECIFIC CAUSATION TESTIMONY FROM PLAINTFF'S EXPERTS

TED STEWART, District Judge.

***1** This matter is before the Court on Defendant Crane Co.'s Renewed Motion to Exclude the Proposed Specific Causation Testimony from Plaintiff's Experts. Defendants York International Corporation, Honeywell, Inc., Goulds Pumps, Flowserve Corporation, and Sepco Corporation (collectively "Defendants") have all joined in Crane Co.'s Renewed Motion. For the reasons discussed below, the Court will grant Defendants' Motion.

I. BACKGROUND

This matter was initially filed in state court by Joseph Alexander Anderson, Jr., and was removed to this Court on September 1, 2006. Plaintiff's complaint alleged that Mr. Anderson had been diagnosed with asbestos-caused Mesothelioma. Mr. Anderson died of Mesothelioma on June 7, 2008, and his wife and the executor of his estate, Arva Anderson, was substituted as Plaintiff. On October 20, 2006, the United States of America Judicial Panel on Multidistrict Litigation issued Conditional Transfer Order 269, [1] which transferred Plaintiff's case to the United States District Court for the Eastern District of Pennsylvania (the "Pennsylvania Court").

On September 26, 2012, without giving any reasoning for its determination, the Pennsylvania Court issued an order denying Defendant Crane Co.'s Motion to Exclude as moot. [2] On the same day, the Pennsylvania Court issued a Suggestion

of Remand, suggesting that the case be remanded to this Court because all discovery had been completed and the case was ready for trial.[3] On October 12, 2012, a Clerk's Order of Conditional Remand was signed, remanding the case back to this Court for trial and severing all claims for punitive or exemplary damages.[4] Soon thereafter, on December 3, 2012, Defendant Crane Co. filed its Renewed Motion to Exclude the Proposed Specific Causation Testimony from Plaintiff's Experts.

Plaintiff hired two experts to testify regarding the cause of Mr. Anderson's Mesothelioma. Drs. Barry Horn and Steven Dikman have each submitted expert reports and have been deposed by Defendants in regard to those reports. Before making their reports, both experts reviewed Mr. Anderson's medical records and work history as supplied by Plaintiff's counsel. However, neither expert personally spoke with or examined Mr. Anderson.

Dr. Dikman passed away on November 8, 2012, and Defendants have withdrawn their arguments as to his personal testimony. However, Defendants have not withdrawn their arguments as to the substance of Dr. Dikman's proposed testimony, and continue to seek an order that no expert should be allowed to offer "every exposure" testimony or give specific causation testimony regarding any of Defendants' products. As the substance of Dr. Dikman's report is at issue in the present Motion, it will be considered despite his death.

A. DR. HORN'S REPORT

Dr. Horn's report consists of a detailed summary of the medical information provided to him, a recitation of Mr. Anderson's work history, and a brief opinion. Dr. Horn opines that "[a]ll of Mr. Anderson's asbestos exposure should be considered a contributing factor in the development of his malignancy. In summary, Mr. Anderson has been diagnosed as having malignant mesothelioma caused by prior occupational and paraoccupational exposure to asbestos."[5] He further opines that "[t]here is only one known cause of malignant mesothelioma in man, and that [is] prior asbestos exposure or exposure to a similar substance called zeolite.... [M]any studies have clearly demonstrated that workers exposed to asbestos are at risk for this otherwise rare malignancy."[6] Dr. Horn later submitted a supplemental report in which he declares that "[t]he mesothelioma was caused by prior exposure to asbestos as outlined in my prior report."[7]

***2** When questioned in his deposition about the basis for his opinions, Dr. Horn affirmed that he "didn't consult anything specific for this case."[8] Furthermore, when asked specifically if he had any opinions related to Defendant Crane Co.'s products, Dr. Horn testified "[n]o, I have no specific opinions. I have no information regarding this man's exposure to Crane Co. Products."[9] Neither Dr. Horn's report nor deposition contains any information regarding Mr. Anderson's exposure to the products of any specific Defendant.

When questioned about whether he needed to know the dose of asbestos dust Mr. Anderson was exposed to in formulating his opinion, Dr. Horn testified "No. If the exposure is above background, then it increased his risk. Now, if there are some exposures that are much higher than other exposures, then the higher exposures would contribute a greater risk than lower exposures. But any exposure above background would increase his risk."[10] He further explained that

> [a]ll chemical carcinogens manifest a dose-dependent relationship. There's, I don't believe there's any dispute anywhere in the literature regarding that issue. The more of a chemical carcinogen you are exposed to, the greater your risk for the development of cancer. This is clearly also true for asbestos; that is, the more asbestos you inhale and retain in your lungs, the greater your risk for developing an asbestos-related disease, and that includes mesothelioma.[11]

B. DR. DIKMAN'S REPORT

Dr. Dikman's report consists of a brief summary of Mr. Anderson's work and medical history followed by a one paragraph opinion. Dr. Dikman opines as follows:

> Asbestos exposure is well documented to cause malignant mesothelioma. The finding of hyalinized pleural plaquing in the surgical tissue specimen from Mr. Anderson indicates asbestos related pleural disease and confirms that his asbestos exposure was substantial. The radiographic and

> clinical findings, including the intraoperative appearance, and the microscopic and immunopathologic studies established the diagnosis of malignant mesothelioma. It is my opinion, with a reasonable degree of medical certainty, that Mr. Anderson's malignant pleural mesothelioma was caused by his asbestos exposure. [12]

When asked whether his opinion on causation required a consideration of the frequency of exposures, Dr. Dikman stated that "[i]n some cases, yes, generally I would say yes but in specifically in Mr. Anderson we have high hyalinized pleural plaquing which documents that he had substantial exposure in the past which would cover both frequency and duration." [13] Dr. Dikman later clarified that he didn't have specific information on Mr. Anderson's exposure to asbestos, stating that he didn't "have specific information as to those types of frequency, duration, and things of that sort." [14]

Additionally, Dr. Dikman testified as follows:

> Q. Did you think that every exposure contributes to the development of this disease?
>
> ***3** A. I would think there is no way of separating every specific exposure, but I would say that, yes, that the aggregate of the exposures did contribute to his disease.
>
> Q. Let me ask you: Do you believe that every exposure contributes to his disease?
>
> A. I would say, yes, and then I have no way of separating one exposure from the other. We know this individual, as many individuals, has had substantial exposure to asbestos and they have an asbestos-caused mesothelioma.
>
> Q. Do you believe that every exposure to asbestos contributes equally to the development of the disease?
>
> A. I don't know.
>
> MR. KIELY: Objection.
>
> Q. Do you believe that the frequency of exposures affects its contribution to the development of disease?
>
> A. We don't know. We don't know the specific threshold of what is needed to develop mesothelioma. There is no established threshold. And the types and amounts of exposure and duration and frequency is very variable, and it's at all different levels. So there is really no specific duration or amount that's really known to be necessary to cause mesothelioma. [15]

Finally, Dr. Dikman testified that the general population is exposed to asbestos in the ambient air. [16] Dr. Dikman clarified that "the measurements in the air samples in the general population, and air samples have been used to see the incidence in a background population from nonexposed individuals, and this has never been shown to have a significant increase in the risk for mesothelioma in these background populations." [17]

II. RULE 702 AND *DAUBERT*

Defendants do not argue that Plaintiff's experts are not qualified to testify as experts in this matter. Instead, Defendants seek to exclude any specific causation testimony that the asbestos which caused Mr. Anderson's disease came from their products. Specifically, Defendants argue that the experts should be precluded from offering testimony that "every exposure" to asbestos is a factual cause of the development of mesothelioma. Defendants argue that such testimony should be excluded because (1) it is not based on sound scientific principles and should be excluded under *Daubert* and Fed.R.Evid. 702; and (2) jurisdictions applying a substantial factor causation test should not permit this type of opinion evidence. As the Court finds that the proposed testimony does not meet the requirements of Rule 702 and *Daubert,* there is no need to consider whether the testimony is appropriate under the substantial factor causation test.

Fed.R.Evid. 702 provides:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:
>
> (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
>
> (b) the testimony is based on sufficient facts or data;
>
> (c) the testimony is the product of reliable principles and methods; and

***4** (d) the expert has reliably applied the principles and methods to the facts of the case.

In *Daubert v. Merrell Dow Pharmaceuticals Inc.* [18] and *Kumho Tire Co., Ltd. v. Carmichael,* [19] the Supreme Court interpreted the requirements of Rule 702. "*Daubert* requires a trial judge to 'ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable." [20] "In applying Rule 702, the trial court has the responsibility of acting as a gatekeeper." [21]

[1] "Scientific knowledge ... 'implies a grounding in the methods and procedures of science' which must be based on actual knowledge and not 'subjective belief or unsupported speculation." [22] "In other words, 'an inference or assertion must be derived by the scientific method ... [and] must be supported by ... good grounds, based on what is known." [23] " 'Under the regime of *Daubert* ... a district judge asked to admit scientific evidence must determine whether the evidence is genuinely scientific, as distinct from being unscientific speculation offered by a genuine scientist." [24]

[2] "The Supreme Court has provided some guidance for the task of determining scientific validity." [25] "This inquiry is 'a flexible one,' not governed by a 'definitive checklist or test.' " [26] Some factors to consider are whether the expert's theory or technique: (1) can be (and has been) tested; (2) has been subjected to peer review and publication; (3) has a known or potential rate of error with standards controlling the technique's operation; and (4) enjoys widespread acceptance in the relevant scientific community. [27]

III. DISCUSSION

Plaintiff's experts admit that they do not have any specific information regarding Mr. Anderson's exposure to any of Defendants' products. [28] They do not appear to think such information is necessary, as their testimony that every exposure Mr. Anderson had to an asbestos fiber contributed to the causation of his disease would imply specific causation regardless of the dose of the exposure or the type of fiber to which Mr. Anderson was exposed. Under this analysis, all Plaintiff must do at trial is show that Mr. Anderson was exposed to some minimal amount of asbestos from the product of a Defendant at some point in his life, and that Defendant could be found liable for his mesothelioma. This would be true regardless of whether or not Mr. Anderson was also exposed to significant amounts of highly carcinogenic fibers from one or more of the other Defendants.

Defendants argue that the testimony of Plaintiff's experts is conjecture that is not based on sound scientific principles or evidence. Therefore, Defendants urge the Court to exercise its gatekeeping powers to exclude this evidence under Rule 702 and *Daubert.* The chief dispute is whether the testimony is based on sufficient facts or data, and whether the testimony is the product of reliable principles and methods.

***5** Recently, in *Smith v. Ford Motor Company,* Judge Dee Benson of this Court thoroughly considered whether Rule 702 and *Daubert* permit expert testimony that "every exposure" to asbestos is a contributing cause to a person's mesothelioma. [29] The Court finds Judge Benson's opinion to be persuasive and well-reasoned. [30] In *Smith,* the plaintiff's expert sought to offer "every exposure" testimony to show that Mr. Smith's mesothelioma was caused by his cumulative exposure to asbestos, with each exposure playing a contributing role, including any exposure he may have had when he changed automobile break pads on several occasions. [31] However, the expert did not have any underlying data on the quantity of fibers found in brakes necessary to cause cancer in a human being. [32]

After thoroughly considering the arguments before it, the Court found the expert opinion to be, "as a matter of law, unsupported by sufficient or reliable scientific research, data, investigations or studies, and is inadmissible under Rule 702." [33] The Court found that "the every exposure theory as offered as a basis for legal liability is inadmissible speculation that is devoid of responsible scientific support." [34]

A. UNDERLYING FACTS AND DATA

Plaintiff's experts are unable to point to any studies showing that "any exposure" to asbestos above the background level of asbestos in the ambient air is causal of mesothelioma. Instead, Plaintiff's experts base their opinion on the fact that scientists have been unable to determine a safe level for exposure to asbestos. Such studies are difficult to perform as mesothelioma often develops as long as between ten and forty years after exposure to asbestos, [35] and scientists have not yet found a way to determine which exposure or fiber(s) caused the mesothelioma. As Dr. Dikman testified, "We don't know.

We don't know the specific threshold of what is needed to develop mesothelioma. There is no established threshold.... So there is really no specific duration or amount that's really known to be necessary to cause mesothelioma." [36]

As noted earlier, not only do Plaintiff's experts lack data on the level of exposure to asbestos necessary to cause mesothelioma, they have no information on Mr. Anderson's exposure to Defendants' products, or even the type of asbestos fibers that Defendants' products may contain. All of the experts' data comes from medical reports which demonstrate that, at some point, Mr. Anderson was exposed to asbestos and that the "asbestos exposure was substantial ." [37] The experts have no information on whether that substantial exposure had any relation to the remaining Defendants before the Court.

[3] As this Court recently stated in *Smith,* "Rule 702 and *Daubert* recognize above all else that to be useful to a jury an expert's opinion must be based on sufficient facts and data. The every exposure theory is based on the opposite: a lack of facts and data." [38] Plaintiffs experts do not base their opinions on scientific evidence that every exposure to asbestos causes mesothelioma. Instead, their testimony is based on their lack of information sufficient to show the level of exposure which does not create a risk of mesothelioma. This is not reliable enough evidence for the Court to allow it in under the standards of *Daubert* and Rule 702. "Just because we cannot rule anything out does not mean we can rule everything in." [39]

B. PRINCIPLES AND METHODS

***6 [4]** "It is well established that a plaintiff in a toxic tort case must prove that he or she was exposed to and injured by a harmful substance manufactured by the defendant." [40] Normally, a plaintiff will rely on its expert to "demonstrate 'the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover. ' " [41] Here, however, Plaintiff's experts simply assert that any level of exposure is hazardous to human beings and forego any examination of Mr. Anderson's actual level of exposure.

In support of his proposed testimony that every exposure to asbestos is casual of mesothelioma, Dr. Horn states that mesothelioma is dose-responsive to asbestos exposure. [42] In addition, Dr. Dikman testified that although there is some background level of asbestos to which the general population is exposed, this exposure has not been shown to pose a significant risk of mesothelioma. [43] The experts simply do not have the scientific information to allow them to testify in further detail regarding a dosage that does pose a significant risk of mesothelioma.

Considering the *Daubert* factors for examining a scientific theory, the theory proposed by Plaintiff's experts is troubling. Due to the significant lag between exposure to asbestos and a diagnosis of mesothelioma, the theory cannot be easily tested. Plaintiff's experts testified that they have no way of knowing which fibers or which exposure caused the mesothelioma. Similarly, there is no known error rate for this theory. Although Plaintiff has pointed to instances in which people with very little known exposure to asbestos contracted mesothelioma, it is not known if the odds of people with so little exposure contracting mesothelioma is one out of a million or one out of a hundred.

Plaintiff has supplemented the record with numerous scholarly articles and scientific studies in support of the claim that asbestos causes mesothelioma and that there is no known safe exposure to asbestos. However, Plaintiff's experts have pointed to no studies showing that the type of exposure Mr. Anderson had to Defendants' products is likely to cause mesothelioma. Viewed in its most favorable light, the literature shows that any exposure to asbestos *could* cause mesothelioma, but no one knows how likely that is.

Plaintiff has also supplied the Court with the reports and testimony of a core group of experts in similar cases where the experts testified that every exposure to asbestos caused a person's mesothelioma. However, the Court must base its opinion on the facts and testimony presented in this case, rather than on the testimony of experts in other cases. Although the testimony of these experts does indicate that the theory has some acceptance in the scientific community, the Court notes that a growing number of courts have determined that the theory is not proper under *Daubert* and Rule 702, expressing the opinion that the " 'any exposure theory is, at most, scientifically-grounded speculation: an untested and potentially untestable hypothesis.' " [44]

***7** For the reasons stated above, the Court finds that the every exposure theory of causation does not meet the standards set by Rule 702 and *Daubert* and must be excluded. Therefore, Defendants' Motion will be granted.

IV. CONCLUSION

It is therefore

ORDERED that Defendants' Renewed Motion to Exclude the Proposed Specific Causation Testimony from Plaintiff's Experts (Docket No. 270) is GRANTED.

Footnotes

1 *See* Docket No. 143.
2 Docket No. 270–8.
3 Docket No. 254.
4 *Id.*
5 Docket No. 270–1 Ex. A, at 12.
6 *Id.* at 10.
7 Docket No. 285–7 Ex. D, at 4.
8 Docket No. 270–2 Ex. 2, at 19.
9 Docket No. 270–3, at 87.
10 Docket No. 270–2 Ex. 2, at 27.
11 *Id.* at 17.
12 Docket No. 285–7 Ex. B, at 2.
13 Docket No. 270–2 Ex. 1, at 13–14.
14 *Id.* at 18–19.
15 *Id.* at 46–48.
16 *Id.* at 126.
17 *Id.* at 128.
18 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993).
19 526 U.S. 137, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999).
20 *Atl. Richfield Co. v. Farm Credit Bank of Wichita,* 226 F.3d 1138, 1163 (10th Cir.2000) (quoting *Daubert,* 509 U.S. at 589).
21 *In re Breast Implant Litig.,* 11 F.Supp.2d 1217, 1222 (D.Colo.1998).
22 *Mitchell v. Gencorp Inc.,* 165 F.3d 778, 780 (10th Cir.1999) (quoting *Daubert,* 509 U.S. at 590).
23 *Id.* (quoting *Daubert,* 509 U.S. at 590).
24 *Id.* at 783 (quoting *Rosen v. Ciba–Geigy Corp.,* 78 F.3d 316, 318 (7th Cir.1996)).
25 *In re Breast Implant Litig.,* 11 F.Supp.2d at 1223.
26 *Atl. Richfield,* 226 F.3d 1163 (quoting *Daubert,* 509 U.S. at 593).
27 *Id.*
28 Docket No. 270–3, at 87; Docket No. 270–2 Ex. 1, at 18–19.
29 2013 WL 214378 (D.Utah Jan.18, 2013).
30 This Court had previously considered whether to allow testimony similar to the testimony proposed here when presented with the question in the context of a motion in limine in *Larson v. Bondex International, Inc.* 2011 U.S. Dist. LEXIS 79830 (D.Utah July 21, 2011). Without the aid of the extensive briefing provided by the parties to the present case, the Court allowed the testimony. *Id.* at *4.
31 *Id.* at *1.
32 *Id.* at *3.
33 *Id.* at *2.
34 *Id.*
35 Docket No. 285–1, at 25.
36 Docket No. 270–2 Ex. 1, at 48.
37 Docket No. 285–7 Ex. B, at 2.
38 2013 WL 214378, at *2.
39 *Id.* at *3.

40 *Mitchell,* 165 F.3d at 781 (citing *Wright v. Willamette Indus., Inc.,* 91 F.3d 1105, 1106 (8th Cir.1996); *Wintz By & Through Wintz v. Northrop Corp.,* 110 F.3d 508, 515 (7th Cir.1997); *Allen v. Pa. Eng'g Corp.,* 102 F.3d 194, 199 (5th Cir.1996)).

41 *Id.* (quoting *Wright,* 91 F.3d at 1106).

42 Docket No. 270–2 Ex. 2, at 17.

43 *Id.* Ex. 1, at 128.

44 *Smith,* 2013 WL 214378, at *5 (quoting *Butler v. Union Carbide Corp.,* 310 Ga.App. 21, 712 S.E.2d 537, 552 (Ga.Ct.App.2011)).

EXHIBIT 8

2012 WL 1570129
Only the Westlaw citation is currently available.
United States District Court,
N.D. Illinois.

SEGLE
v.
STEGMILLER.

No. 10 C 4618. | May 3, 2012.

Attorneys and Law Firms

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Opinion

STATEMENT

HARRY D. LEINENWEBER, Judge.

I. Background

***1** Plaintiff has moved to bar testimony from Defendants' expert Dr. Paul Akers. Dr. Akers is, according to Defendants, an experienced maxillofacial surgeon and diplomat to the American Board of Oral & Maxillofacial Surgery. Although a copy of his resume, publications, and previous testimony has not been provided to the Court, Defendants aver (and Plaintiff does not dispute) that that information has been provided to Plaintiff. In any event, Plaintiff challenges the adequacy of Akers' report, not his qualifications.

The substantive portion of Dr. Akers' expert opinion is a one-page letter. In it, Dr. Akers states that "it is my medical opinion, that within a reasonable degree of certainty that [Plaintiff's] broken jaw may have occurred while [Defendants subdued] Mr. Sigle and forc[ed] him to the ground while he was resisting arrest. The fracture may have occurred when Mr. Sigle hit the floor in the hallway." Essentially, Dr. Akers concludes that the defendants' account is "very plausible" in light of the injuries as described in the medical records, but that the relative lack of facial bruising or lacerations is, in his experience as a maxillofacial surgeon, inconsistent with being hit or kicked multiple times in the face. Accordingly, Dr. Akers concluded that Mr. Sigle's version of events was inconsistent with the medical evidence that he reviewed.

II. Legal standard

Rule 26 requires a party to disclose "a complete statement of all opinions [an expert witness]will express and the basis and reasons for them." Rule 26(a)(2)(B)(i). Whether such testimony is admissible is governed by Federal Rule of Evidence 702, and the line of cases originating with *Daubert v. Merrell Dow Pharms., Inc.,* 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993). *See Ervin v. Johnson & Johnson, Inc.,* 492 F.3d 901, 904 (7th Cir.2007). Courts undertake a three-step analysis to determine whether such testimony is relevant and reliable, not "unscientific speculation offered by a genuine scientist." *Rosen v. Ciba–Geigy Corp.,* 78 F.3d 316, 318 (7th Cir.1996). First, the witness must be qualified as an expert based on his knowledge, skill, experience, training, or education. Fed.R.Evid. 702. Second, his reasoning or methodology must be scientifically reliable. *Ervin,* 492 F.3d at 904. Courts consider a variety of factors in determining whether an opinion is reliable, including whether the theory has been tested or published, and whether it is generally accepted in the scientific community; however, the inquiry is flexible and relevant factors vary by the kind of expertise at issue. *See Smith v. Ford Motor Co.,* 215 F.3d 713, 719–20 (7th Cir.2000). Finally, the testimony must assist the trier of fact to understand certain evidence or determine a fact at issue in the case. *Ervin,* 492 F.3d at 904.

III. Discussion

Plaintiff objects that the expert report does not satisfy Rule 26 in that it is unsigned and insufficiently sets forth the basis for Akers' conclusions. There is no dispute that the report is unsigned, but Plaintiff has identified no prejudice that he suffered by that error. It was harmless. The Court accordingly permits Defendants to produce a corrected copy of the expert report which includes a proper signature.

***2** As to the substance of the report, the Court notes that the substantive portion of Dr. Akers' report is hardly overwhelming in its detail. Nonetheless, Dr. Akers notes that he has reviewed the medical records from two relevant hospitals, transcripts of IPRA investigations, and the parties' depositions. Although he sets forth Defendants' account at

greater length than Plaintiff's, Dr. Akers notes the conflicting stories and states that in his medical experience, Defendants' account of what transpired is a plausible explanation for Plaintiff's injuries, whereas Plaintiff's account is not. *Cf. Mosby v. Silberschmidt,* No. 08–cv–677, 2010 WL 4536999, at *2 (W.D.Wis. Nov.2, 2010) (noting that where the expert explained what documents he reviewed and applied his experience as a practicing dentist to that information, the brevity of the expert report did not render it inadmissible). The fact that Dr. Akers did not examine Plaintiff before offering his opinion does not render that opinion unreliable or otherwise impermissible. *See Walker v. Soo Line R. Co.,* 208 F.3d 581, 591 (7th Cir.2000). Accordingly, the Court does not agree that Dr. Akers' opinion is unreliable in that it fails to provide a basis for his conclusions, or merely parrots Defendants' story.

The shortness of the opinion is doubtless attributable at least in part to its simplicity: Akers' opinion is essentially that any significant blunt trauma to the face can result in a broken jaw, and that both parties have proffered a theory of what that trauma was in this case. Based on Dr. Akers' medical experience, the absence of noted significant facial lacerations or bruising makes Plaintiff's account of the incident inconsistent with the medical evidence. Plaintiff objects that Akers is not qualified as an expert in how jaws are fractured, but he doesn't have to be. Nowhere in his report does he state that he knows what caused the fracture—he merely opines that Defendants' account is consistent with a force that could have broken Plaintiff's jaw (and unlike Plaintiff's version, is evidently not undermined by other medical evidence). It is well understood that blunt trauma can result in a jaw fracture, and that punches and kicks to the face generally produce cuts and bruises—almost to the point of not requiring expert testimony. They certainly do not require any more expertise than Dr. Akers has.

It is a closer question, however, whether Dr. Akers' opinions will be helpful to the jury. If it were true, as Plaintiff's briefing implies, that Akers gave no more than his general conclusion that Defendants' account could have produced Plaintiff's broken jaw, this Court would be inclined to exclude that opinion as too general to be helpful. *Cf. Myers v. Illinois Central R. Co.,* 629 F.3d 639, 644 (7th Cir.2010) (conducting a reliable differential etiology involves ruling possible causes of a condition in or out). Unlike in *Myers,* and despite Plaintiff's careful avoidance of this point, Dr. Akers did rule something out—Plaintiff's explanation of how he was injured. *See White v. City of Chicago,* No. 07 C 2539, 2011 WL 679905, at * 11 (N.D.Ill. Feb.16, 2011) (A doctor may testify that his professional experience is inconsistent with a plaintiff's account of injuries). To the extent that his medical experience allows him to rule out Plaintiff's explanation, as set forth in Plaintiff's own deposition, he has adequately stated the basis for his conclusion and the testimony could help the jury resolve the contested issue of fact. *Cf. Banister v. Burton,* 636 F.3d 828, 832 (7th Cir.2011) (treating physician gave descriptive testimony and then applied his basic medical knowledge to that information in opining that a plaintiff's injuries would not have stopped him from moving in certain ways.).

***3** It is not, however, particularly hard for a layperson to grasp that a fractured jaw can be caused by blunt trauma, or that a significant beating to the face would be expected to result in bleeding, swelling, or lacerations. *See Dhillon v. Crown Controls Corp.,* 269 F.3d 865, 871 (7th Cir.2001) (to be helpful to the jury, an expert must testify to something that is not obvious to a layman). Nonetheless, because the individual experiences of prospective jurors may vary considerably, the Court does not conclude that the proffered opinion is so obvious that brief expert testimony would be impermissible under the circumstances. Given that there is no dispute that Dr. Akers is qualified, and that this Court has found his opinions to be sufficiently reliable and helpful to the jury, Plaintiff's motion to bar is denied.

The Court notes, however, that Defendants will be held to the thinness of the report. For example, it contains no statement that there is anything unique to Plaintiff's jaw fracture that is especially consistent with Defendants' story. Defendants will not be permitted later to offer opinions not disclosed in the report.

IV. Conclusion

For the foregoing reasons, the Court denies Plaintiff's ocean to bar the testimony of Defense expert Dr. Akers.

End of Document

EXHIBIT 9

2011 WL 3207363
Only the Westlaw citation is currently available.
United States District Court,
S.D. Ohio,
Western Division.

Susan CLEMENTS–JEFFREY, et al., Plaintiffs,
v.
CITY OF SPRINGFIELD, OHIO, et al., Defendants.

No. 3:09–cv–84. | July 27, 2011.

Attorneys and Law Firms

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Opinion

DECISION AND ENTRY SUSTAINING PLAINTIFF'S MOTION IN LIMINE TO EXCLUDE TESTIMONY OF DR. ARTHUR J. JIPSON (DOC. # 93)

WALTER HERBERT RICE, District Judge.

***1** Plaintiffs have moved the Court for an Order prohibiting Defendants from introducing, at trial, the testimony of Defendants' expert witness, Dr. Arthur J. Jipson, an Associate Professor of Sociology and Director of the Criminal Justice Studies Program at the University of Dayton. For the reasons stated below, the Court sustains Plaintiffs' motion (Doc. # 93). [1]

I. Dr. Jipson's Proposed Testimony

Defendants offer Dr. Jipson as a "contextual expert," who can help to explain the problems of laptop computer theft, the need for theft recovery tools, and how those tools operate. He also intends to offer his expert opinion that Plaintiffs had no reasonable expectation of privacy in communications via the Internet.

Dr. Jipson offers five conclusions in his expert report:

1. It is not reasonable to believe that electronic communication is private online.

2. Only the original owner of a computer can have meaningful knowledge of security protection it contains. Any subsequent user of a laptop cannot assume automatic protection of any kind.

3. Computer, laptop, and electronic equipment theft is a serious social and criminological problem for organizations, businesses and individuals that requires reasonable remote and location-specific security solutions.

4. When a company activates system operation software capture for security reasons, the representatives of the company/ employees cannot predict the nature of the material that will be accessed.

5. Security and theft protection tools are necessary and proper tools to combat computer theft.

Ex. 1 to Jipson Decl. attached to Absolute Defs.' Mot. for Summ. J.

II. Federal Rule of Evidence 702

Plaintiffs maintain that Dr. Jipson's testimony does not satisfy the standards for expert witness testimony under Federal Rule of Evidence 702. That Rule states:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed.R.Evid. 702.

In *Daubert v. Merrell Dow Pharmaceuticals, Inc.,* 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993), the Supreme

Court held that the trial judge must perform a "gatekeeping" function with respect to expert witness testimony. *Id.* at 596. The court must ensure that expert witness testimony "is not only relevant, but reliable." *Id.* at 589.

With respect to relevance, the question is whether the expert testimony being proffered "is sufficiently tied to the facts of the case that it will aid the jury in resolving a factual dispute." *Id. at 591 (quoting United States v. Downing,* 753 F.2d 1224, 1242 (3d Cir.1985)). With respect to the question of reliability, where the expert's testimony is based on something other than scientific knowledge, the court is given "broad latitude" in determining whether the proffered testimony is sufficiently reliable. *See Kumho Tire Co., Ltd. v. Carmichael,* 526 U.S. 137, 141–42, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999).

III. Qualifications

***2** The Court turns first to the question of Dr. Jipson's qualifications. The parties disagree on whether Dr. Jipson is qualified, by virtue of his "knowledge, skill, experience, training, or education," to offer expert witness testimony on the topics of Internet privacy, laptop computer theft, and theft recovery tools. *See* Fed.R.Evid. 702.

Defendants note that Dr. Jipson has taught classes in sociology and criminology at the university level for almost 20 years. He teaches classes on cyber crime and Internet deviance. Internet crime and privacy issues have been a consistent area of interest for him. He teaches a course on Internet and Popular Culture, which includes a section on Internet privacy. He has also written numerous articles that touch on these topics. Plaintiffs note, however, that none of these articles deals exclusively with the subject of his opinions in this case, and that he has never testified as an expert witness on this topic. Jipson Dep. at 10–12, 33–34, 48, 56–58, 62.

The Court need not decide whether Dr. Jipson possesses the requisite qualifications to offer the proposed expert witness testimony. Assuming *arguendo* that he is qualified, his testimony is nevertheless inadmissible for other reasons.

IV. Whether Plaintiffs Had a Reasonable Expectation of Privacy Is a Question of Law and, Given that Dr. Jipson's Opinion Is Contrary to Law, His Opinion on this Subject is Not Relevant to the Issues in this Case.

Based on his knowledge, education, and experience, Dr. Jipson first offers his expert opinion that no one, including Plaintiffs, has a reasonable expectation of privacy in Internet communications. As will be noted in the Decision and Entry ruling on Defendants' motions for summary judgment, this is a threshold issue in this case, and a necessary prerequisite for each of Plaintiffs' claims. *See United States v. Jones,* 75 F. App'x 398, 400 (6th Cir.2003) (noting that a reasonable expectation of privacy is "tantamount to 'standing' in other contexts").

The question of whether Plaintiffs had a reasonable expectation of privacy in their Internet communications, however, is a question of law to be decided by the Court. *See id.; United States v. Welliver,* 976 F.2d 1148, 1151 (8th Cir.1992). This renders Dr. Jipson's "opinion" on this topic absolutely irrelevant. What makes his "opinion" even more troublesome is that it is contrary to case law.

Numerous courts have recognized that individuals have an objectively reasonable expectation of privacy in their personal computers. *See United States v. Heckenkamp,* 482 F.3d 1142, 1146 (9th Cir.2007) (holding that the defendant "had a legitimate, objectively reasonable expectation of privacy in his personal computer"); *United States v. Lifshitz,* 369 F.3d 173, 190 (2d Cir.2004) ("Individuals generally possess a reasonable expectation of privacy in their home computers."); *Guest v. Leis,* 255 F.3d 325, 333 (6th Cir.2001) ("Home owners would of course have a reasonable expectation of privacy in their homes and in their belongings-including computers-inside the home.").

***3** Personal computers that are password-protected are subject to even greater privacy protection. *See United States v. Aaron,* 33 F. App'x 180 (6th Cir.2002) (in assessing the scope of a privacy interest, the court should examine "whether the relevant files were password-protected or whether the defendant otherwise manifested an intention to restrict third-party access."); *United States v. Lucas,* 640 F.3d 168 (6th Cir.2011) (holding that the district court did not err in holding that the search of a laptop computer that was not password-protected was akin to the search of a closed, unlocked container); *United States v. Buckner,* 473 F.3d 551, 554 n. 2 (4th Cir.2007) (district court's finding that defendant had a reasonable expectation of privacy in password-protected files was not clearly erroneous).

As to electronic communications sent over the Internet, the Sixth Circuit has recently held that "a subscriber enjoys

a reasonable expectation of privacy in the contents of emails 'that are stored with, or sent or received through, a commercial [Internet service provider].' " *United States v. Warshak,* 631 F.3d 266, 288 (6th Cir.2010), reh'g and reh'g en banc denied (2011) (quoting *Warshak v. United States,* 490 F.3d 455, 473 (6th Cir.2007)). The court found that "the very fact that information is being passed through a communications network is a paramount Fourth Amendment consideration." *Id.* at 285. It also stated that "the Fourth Amendment must keep pace with the inexorable march of technological progress, or its guarantees will wither and perish." *Id.* The Sixth Circuit noted that Fourth Amendment protects traditional forms of communications such as telephone calls and letters, and found that "it would defy common sense to afford emails lesser Fourth Amendment protection." *Id.* at 286.

The court in *Warshak* also held that even though email had to pass through an Internet service provider ("ISP"), and even though that provider may have contractually reserved the right to access the subscriber's email in certain circumstances, neither the ability of the ISP to gain that access, nor its contractual right to do so, extinguished the user's reasonable expectation of privacy. *Id.* at 286–87.

In a similar vein, the Supreme Court recently assumed, without deciding, that a city employee had a reasonable expectation of privacy in text messages sent and received on a pager provided by his employer. *See City of Ontario v. Quon,* —— U.S. ——, ——, 130 S.Ct. 2619, 2630, 177 L.Ed.2d 216 (2010).

These holdings can logically be extended to cover instant messages and webcam communications, the types of electronic communications at issue in this case. Applicable statutes also shed light on whether an individual has an objectively reasonable expectation of privacy in electronic communications. The Stored Communications Act ("SCA"), 18 U.S.C. § 1701 *et seq.,* at issue in *Warshak* and *Quon* and the subject of one of Plaintiffs' claims in this case, specifically prohibits the intentional, unauthorized access of stored communications such as email. The Electronic Communications Privacy Act ("ECPA"), 18 U.S.C. § 2511, also the subject of one of Plaintiffs' claims in this case, specifically prohibits the intentional, unauthorized interception, disclosure, and use of wire, oral, and electronic communications.

***4** Based on these statutes and on the above-cited case law, the Court concludes that Dr. Jipson's expert "opinion," that no one has an objectively reasonable expectation of privacy in password-protected Internet communications, is contrary to law, and thus not relevant to the issues in this litigation.

The Court finds it curious that despite Dr. Jipson's broadly stated expert opinion-that there is no reasonable expectation of privacy in communications via the Internet–Defendants did not argue this in their motions for summary judgment. Rather, they argued only that Plaintiffs lacked an objectively reasonable expectation of privacy because they knew or should have known that the laptop computer being used by Clements–Jeffrey was stolen. [2] The Court also finds it curious that, in formulating his opinion, Dr. Jipson did not consider this fact at all. Nor did he take the statutory prohibitions set forth in the ECPA and the SCA into account. Jipson Dep. at 81–82, 85–86, 135–36. As Plaintiffs note, Dr. Jipson completely ignored these "core issues."

In any event, the question of whether Plaintiffs had an objectively reasonable expectation of privacy in their Internet communications is a question of law to be determined by the Court. This renders Dr. Jipson's opinion on this topic completely irrelevant, even more so in light of the fact that it is contrary to case law. For these reasons, the Court finds that his opinion on this topic is inadmissible. [3]

V. Even Though the Remainder of Dr. Jipson's Proposed Expert Witness Testimony May Assist the Jury In Understanding Some of the Evidence, It Is Excludable Under Federal Rule of Evidence 403.

Dr. Jipson also intends to offer expert witness testimony concerning the pervasive problem of laptop computer theft and the need for theft recovery tools. In addition, he intends to explain to the jury that theft recovery tools are often present on laptop computers, and when those theft recovery tools are activated, it is difficult to predict the nature of the material that will be accessed. [4] Defendants argue that because these topics are beyond the actual knowledge and expertise of jurors, Dr. Jipson's testimony will assist the jury in its understanding of the relevant subject matter.

In addition to rendering an expert "opinion" on a topic, an expert witness may also be permitted to provide background information on a particular topic if it will assist the jury in understanding a particular issue. 4 Jack Weinstein & Margaret Berger, *Weinstein on Evidence* § 702.02[2] (2d

ed.2006); *United States v. Mulder,* 272 F.3d 91, 102 (2d Cir.2001) ("The government is free to offer expert testimony both as background for an offense and to assist in proving one or more elements of the offense.").

Even though expert witness testimony may be relevant and may assist the jury in understanding the issues, it is nevertheless subject to the balancing test set forth in Federal Rule of Evidence 403. Rule 403 provides that, "[a]lthough relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence."

***5** Plaintiffs maintain that whatever little probative value the remainder of Dr. Jipson's testimony may have, it is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury. The Court agrees. In assessing the probative value of certain testimony, a court may consider what other evidentiary alternatives are available. If alternatives are readily available that have the same or greater probative value, but a lower danger of unfair prejudice or confusion of the issues, the court may exclude testimony on that basis. *See Old Chief v. United States,* 519 U.S. 172, 183–85, 117 S.Ct. 644, 136 L.Ed.2d 574 (1997).

In this case, the Court presumes that Defendant police officers Geoffrey Ashworth and Neil Lopez, and Absolute Software's theft recovery officer Kyle Magnus will called to testify at trial. They will likely testify about the high number of laptop computers that are reported stolen, and about how theft recovery tools may be used to assist law enforcement officials in tracing stolen laptops.

Dr. Jipson testified that he was generally familiar with the operation of various theft recovery tools, including Absolute Software's "LoJack for Laptops," the theft recovery system that was used in this case. Jipson Dep. at 46. However, in the Court's view, Magnus and other Absolute Software employees are in a much better position to describe to a jury how the LoJack system works. They are also in a much better position to explain the various remote access tools used to trace the stolen laptop. In the Court's view, the testimony of these witnesses has significantly greater probative value than that of Dr. Jipson. The Court therefore concludes that the probative value of the remainder of Dr. Jipson's testimony is very slight.

Moreover, Magnus and other Absolute employees will undoubtedly testify about the prevalence of laptop computer theft, theft recovery tools in general, and about LoJack for Laptops in particular. They will also undoubtedly testify about how difficult it is to predict the nature of material that will be accessed when using certain theft recovery tools. Therefore, Dr. Jipson's testimony on these same topics would amount to "needless presentation of cumulative evidence."

In light of the opinions expressed in Dr. Jipson's expert report, there are also significant risks in allowing him to present "expert" testimony concerning these issues. Dr. Jipson believes that no one has any objectively reasonable expectation of privacy in Internet communications, an opinion the Court has found to be contrary to law, and thus inadmissible at trial. Given the likelihood that his belief would creep into his testimony on these other topics, there is a danger that the jury might be misled or confused.

Dr. Jipson is also of the opinion that theft recovery tools, presumably like those used in this case, are "necessary and proper" for combatting the problem of computer theft. Yet, in formulating his opinions, he admits that he completely failed to consider the statutory prohibitions set forth in the ECPA and SCA. This significantly increases the risk that the jury will be misled by his testimony. If the jury determines that Plaintiffs neither knew nor should have known that the laptop computer was stolen, the jury will then be called upon to determine whether the Absolute Defendants' efforts to recover the stolen laptop violated the ECPA or SCA, or otherwise invaded Plaintiffs' protected privacy interests. Dr. Jipson's opinions completely ignore these core issues.

***6** Plaintiffs argue that a jury might give Dr. Jipson's "expert" witness testimony undue weight. They further argue that the admission of his testimony might invite jury nullification. Expert witness testimony that Plaintiffs' beliefs in privacy were unwarranted and that law enforcement should be given significant leeway in recovering stolen laptops could invite the jury to find that the ends justified the means, regardless of whether Defendants' tactics violated the law or invaded Plaintiffs' privacy rights. In the Court's view, Plaintiffs' concerns are not unfounded.

The Court concludes that the slight probative value of Dr. Jipson's testimony on the remainder of the issues is substantially outweighed by the danger of confusion of the issues and misleading the jury. Quite simply, other witnesses are better equipped to provide the same "background"

information to the jury, and can do so without the risks discussed above. Therefore, although relevant, Dr. Jipson's testimony on these topics is inadmissible under Federal Rule of Evidence 403.

VI. Conclusion

For the reasons set forth above, the Court SUSTAINS Plaintiffs' Motion in Limine to Exclude Testimony of Dr. Arthur J. Jipson (Doc. # 93).

Parallel Citations

85 Fed. R. Evid. Serv. 1281

Footnotes

1 Defendants argue that Plaintiffs' motion is not yet ripe because the Court has not yet ruled on Defendants' motions for summary judgment. A separate Decision and Entry ruling on those motions, however, will be issued within a few days, and counsel have already been orally advised as to the Court's ruling therein.

2 As discussed in the Decision and Entry ruling on those motions for summary judgment, the question of whether Plaintiffs knew or should have known that the laptop was stolen is a factual dispute that must be resolved by a jury.

3 Having found that Dr. Jipson's testimony on this topic is irrelevant, the Court need not address the question of whether it is reliable.

4 To the extent that Defendants seek to introduce this particular testimony to support their claim that Plaintiffs had no reasonable expectation of privacy in their Internet communications, it is irrelevant for the reasons previously discussed.

EXHIBIT 10

2010 WL 3893601
Only the Westlaw citation is currently available.
United States District Court,
D. Minnesota.

Mark ANDERSON and Killer Whale Holdings, LLC, a Minnesota limited liability company, Plaintiffs,
v.
DAIRY FARMERS OF AMERICA, INC., a foreign corporation, Defendant.

Civil No. 08–4726 (JRT/FLN). | Sept. 30, 2010.

Attorneys and Law Firms

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Opinion

MEMORANDUM OPINION AND ORDER

JOHN R. TUNHEIM, District Judge.

***1** Plaintiffs Mark Anderson and Killer Whale Holdings, LLC (collectively, "plaintiffs") brought this action against defendant Dairy Farmers of America, Inc. ("DFA"), alleging that DFA violated Section 9 of the Commodity Exchange Act ("CEA") by manipulating prices for cheese and Class III milk futures on the Chicago Mercantile Exchange ("CME"). DFA filed a motion for summary judgment, arguing that plaintiffs have not pleaded or adduced evidence showing that artificial prices for cheese or Class III milk futures existed or that DFA intended to cause artificial prices. Plaintiffs and DFA also filed motions to exclude expert opinions. For the reasons set forth below, the Court denies DFA's motion for summary judgment, grants plaintiffs' motion to exclude James Jordan's expert opinion on mitigation of damages, denies without prejudice DFA's motion to exclude the expert testimony of Wayne Brown, and denies without prejudice plaintiffs' motion to exclude the expert testimony of Robert Mackay.

BACKGROUND [1]

Mark Anderson is a commodities trader who began trading on the CME in late 2001 through his personal account and also through Killer Whale Holdings, LLC, which was formed in 2003. (Anderson Decl. ¶ 11, Docket No. 75.) Plaintiffs traded a variety of commodities on the CME, including Class III milk futures. (*Id.*) DFA is a dairy marketing cooperative owned by 18,000 dairy farmers in 48 states. (Anderson Decl. Ex. C at 2, Docket No. 95.) "DFA markets the milk produced by its members, manufactures dairy products, food components and ingredients, and formulates and packages shelf-stable dairy products." (*Id.*) DFA trades on the CME. (*Id.* at 3.)

I. CLASS III MILK FUTURES TRADING AND THE CHEESE SPOT CALL ON THE CME

Traders meet on the CME Spot Call to trade various futures contracts, including Class III milk futures and cheddar cheese. (Anderson Decl. ¶ 12, Docket No. 75; Harty Decl. ¶ 4, Docket No. 52.) Class III milk futures are traded daily on the CME in units of 200,000 pounds, or 2,000 hundredweight of milk. (Anderson Decl. Ex. C at 2, Docket No. 75.) A Class III milk futures contract is cash-settled against the United States Department of Agriculture Class III milk price. (*Id.*) Class III milk is the milk that is used to make cheese, and the Class III milk prices are calculated by reference to, *inter alia,* the price of cheddar cheese. (*Id.* ¶ 12 & Ex. C at 2.) In other words, cheese prices are a component of the formula that determines the price of Class III fluid milk and Class III milk futures contracts. (*Id.* Ex. C. at 2; Garrod Decl. Ex. A at 22–28, Docket No. 77.)

Cheddar cheese is also offered on the CME Cheese Spot Call in the form of 500–pound barrels and 40–pound blocks, and is traded in 40,000–44,000 pound quantities known as "loads" or "carloads." (Anderson Decl. Ex. C at 2, Docket No. 75; Garrod Decl. Ex. B, Docket No. 77.) The CME cheese spot market is a "thin market"—that is, it handles a very small proportion of all United States bulk cheddar cheese transactions—but the CME cheese spot market effectively sets the market price for most cheese and milk sales across the country. (Garrod Decl. Ex. A. at 9, 22–28, Docket No. 77; *see also* Anderson Decl. Ex. C at 3, Docket No. 75 ("The volume of cheddar cheese traded on the CME Cheese Spot Call comprises less than two percent of the annual U.S. supply of cheddar cheese.").) Unlike futures contracts, such as the

Class III milk futures contracts traded on the CME, "where delivery of the underlying cash product is optional," delivery of cheese traded on the CME Cheese Spot Call occurs within a few business days of the execution of the sale. (Harty Decl. ¶ 4, Docket No. 52.)

II. DFA'S TRADING ACTIVITIES ON THE CME FROM MAY 21, 2004, TO JUNE 22, 2004

***2** Plaintiffs allege that DFA's trading activities from May 21, 2004, through June 22, 2004, (the "relevant time period") violated Section 9 of the CEA, which prohibits the manipulation of prices for commodities in interstate commerce. (*See* Am. Compl. ¶ 18, Docket No. 83.)

In May 2004, plaintiffs—acting on the belief that cheddar cheese prices and Class III milk futures prices would fall—acquired a substantial short position in Class III milk futures contracts that would settle in June, July, and August 2004. (Anderson Decl. ¶¶ 14–16, Docket No. 75.) Although block cheddar cheese prices were $2 .20 per pound in the middle of April 2004, those prices declined slowly, settling at $2.15 through May 11, 2004. (Garrod Decl. Ex. C at DFAI0095589–629 to–634, Docket No. 77.) On May 12, the block cheddar cheese price dropped to $2.00, where it remained for six days. (*Id.* at DFAI0095589–634 to 38.) On May 21, the CME block cheddar cheese price dropped an additional 20 cents to $1.80. (*Id* . at DFAI0095589 to 638.) Between May 21, 2004, and June 22, 2004, the CME block cheddar cheese price remained at $1.80. (Am. Compl. ¶ 22, Docket No. 83.)

In December 2008, the Commodities Futures Trading Commission ("CFTC"), in a proceeding relating to DFA's spring 2004 trading activities on the CME, found:

> Beginning on April 14, 2004, as sellers offered cheddar blocks on the CME Cheese Spot Call, DFA purchased block cheddar cheese. From May 21 to June 23, 2004, DFA ... purchased and took delivery of a total of 323 loads (approximately 40,000 pounds per load) of cheddar cheese blocks at $1.80 per pound on the CME Cheese Spot Call. During this period, DFA was the sole purchaser of cheddar cheese blocks on the CME.

(Anderson Decl. Ex. C at 3, Docket No. 75.)

Notably, in the months leading up to May 2004, DFA had purchased a number of long speculative June, July, and August 2004 Class III milk futures contracts on the CME. (Wilson CFTC Dep. Tr. 47–52, Aug. 3, 2006, Garrod Decl. Ex. D, Docket No. 77.) Plaintiffs allege that DFA held long Class III milk futures contracts in excess of the CME limit of 1500 contracts. *See* CME Rule 5202.E ("No person shall own or control more than: 1500 contracts long or short in any contract month."). Specifically, plaintiffs claim that as of May 21, 2004, DFA and its affiliates held 6,172 June contracts, 4,656 July contracts, and 4,227 August contracts. (Anderson Decl. Ex. C at 3, Docket No. 75.) Because dairy product prices, including cheddar cheese prices, continued to decline, "DFA's Class III milk futures position reflected an unrealized loss." (*Id.*)

Plaintiffs allege that DFA had no need for the cheddar cheese it purchased during the relevant time period. Plaintiffs allege that DFA's purchase of cheddar cheese blocks was an attempt to sustain cheddar cheese and Class III milk futures prices while DFA liquidated its long June, July, and August Class III milk futures contracts. (*See* Garrod Decl. Ex. F, Docket No. 77; *see also* Am. Compl. ¶ 22, Docket No. 83.) Plaintiffs allege that DFA's cheddar cheese purchases in fact supported the cheddar cheese and Class III milk futures prices, which eroded plaintiffs' short June, July, and August 2004 Class III milk futures contracts. (*Id.* ¶¶ 27–28.) Plaintiffs claim that they suffered a combined $6 million loss as a result of DFA's trading activities on the CME Cheese Spot Call during the relevant time period. (*Id.* ¶ 29.) In a letter to the CFTC, DFA stated:

> ***3** It appears that from sometime in May 2004 forward, DFA's primary reason for purchasing block cheese on the CME was to defend the price at $1.80 to protect the value of DFA's existing inventories of physical cheese and its Class III milk futures contract positions. Another effect of supporting the price of cheese was support for the Class III milk price, which directly impacted milk payments to DFA's dairy farmer members. [2]

(Garrod Decl. Ex. 1 at 10–11, Docket No. 124.)

On November 2, 2009, DFA brought a motion for summary judgment arguing that plaintiffs have not established that artificial prices existed for cheese or Class III milk or that DFA intended to cause artificial prices for cheese or Class III milk. DFA also brought a motion to exclude plaintiffs' expert, Wayne Brown. Plaintiffs filed motions to exclude DFA expert Robert Mackay and to exclude DFA expert James Jordan's opinion on mitigation of damages. The Court first addresses DFA's motion for summary judgment, and then turns to the parties' motions to exclude.

I. MOTION FOR SUMMARY JUDGMENT

A. Standard of Review

Summary judgment is appropriate where there are no genuine issues of material fact and the moving party can demonstrate that it is entitled to judgment as a matter of law. Fed.R.Civ.P. 56(c). A fact is material if it might affect the outcome of the suit, and a dispute is genuine if the evidence is such that it could lead a reasonable jury to return a verdict for either party. *Anderson v. Liberty Lobby, Inc.,* 477 U.S. 242, 247 (1986). A court considering a motion for summary judgment must view the facts in the light most favorable to the non-moving party and give that party the benefit of all reasonable inferences that can be drawn from those facts. *Matsushita Elec. Indus. Co. v. Zenith Radio Corp.,* 475 U.S. 574, 587 (1986).

B. CEA Manipulation Claim

Section 9(a)(2) of the CEA makes it unlawful for "[a]ny person to manipulate or attempt to manipulate the price of any commodity in interstate commerce." 7 U.S.C. § 13(a)(2). The CEA does not define the term "manipulate," but federal courts and the CFTC have held that manipulation is "an intentional exaction of a price determined by forces other than supply and demand. *Frey v. CFTC,* 931 F.2d 1171, 1175 (7th Cir.1991); *see also Volkart Bros ., Inc. v. Freeman,* 311 F.2d 52, 58 (5th Cir.1962) ("Manipulation is[ ] any and every operation or transaction or practice ... calculated to produce a price distortion of any kind in any market either in itself or in relation to other markets.... Any and every operation, transaction (or) device, employed to produce these abnormalities of price relationship in futures markets, is manipulation." (third alteration in original) (internal quotation marks omitted)). To establish a market manipulation claim under Section 9(a) of the CEA, the plaintiff must show "(1) the defendant possessed an ability to influence market prices; (2) an artificial price existed; (3) the defendant caused the artificial price; and (4) the defendant specifically intended to cause the artificial price." *In re Amaranth Natural Gas Commodities Litig.,* 587 F.Supp.2d 513, 530 (S.D.N.Y.2008); *accord Hershey v. Energy Transfer Partners, L.P.,* 610 F.3d 239, 247 (5th Cir.2010).

***4** While DFA concedes plaintiffs' factual allegations for the purposes of the motion for summary judgment, including the allegation that DFA intended to influence the price of cheese and Class III milk futures, DFA contends that "[p]laintiffs ... do not allege, nor do they have evidence to show, that DFA intended to or actually created an artificial price for cheese or milk futures. Without proof of artificial price, there can be no manipulation under the CEA." (Def.'s Mem. in Supp. of Mot. for Summ. J. at 9, Docket No. 103.)

1. Artificial Price and Intent to Cause an Artificial Price

As alluded to by DFA, the issue before the Court is whether plaintiffs have pleaded or adduced evidence that an artificial price for cheese and Class III milk futures existed.[3] An artificial price is a "price which does not reflect basic forces of supply and demand." *Cargill, Inc. v. Hardin,* 452 F.2d 1154, 1163 (8th Cir.1971); *see also United States v. Socony–Vacuum Oil Co.,* 310 U.S. 150, 223 (1940) ("[M]arket manipulation in its various manifestations is implicitly an artificial stimulus applied to ... market prices, a force which distorts those prices, a factor which prevents the determination of those prices by free competition alone."). To determine whether a price is artificial,

> One must look to the aggregate forces of supply and demand and search for those factors which ... are not a legitimate part of the economic pricing of the commodity.... [W]hen a price is effected by a factor which is not legitimate, the resulting price is necessarily artificial. Thus, the focus should not be as much on the ultimate price, as on the nature of the factors causing it.
>
> *In re Ind. Farm Bureau Coop. Ass'n,* [1982–1984 Transfer Binder] Comm. Fut. Law. Rep. (CCH) ¶ 21,796, at 27,288 n. 2; *accord CFTC v. Enron Corp.,* Civ. No. H–03–909, 2004 WL 594752, at *6 (S.D. Tex Mar. 10, 2004).

DFA asserts that "[i]n order to be manipulative, one must commit fraud, engage in sham or otherwise fictitious transactions or cornering activity, or trade in a manner that disrupts orderly trading processes or violates applicable trading rules." (Def.'s Mem. in Supp. of Mot. for Summ. J. at 20, Docket No. 103.) DFA contends that evidence of its intent to influence price, without establishing fraud, misrepresentation, or violation of trading rules, is insufficient

to establish a CEA manipulation claim and that DFA's cheese purchases were a legitimate part of supply and demand. The Court disagrees and declines to adopt or apply DFA's proposed standard.

Courts and the CFTC "generally agree that manipulation defies easy description. As a result, manipulation cases tend to be characterized by fact-specific, case-by-case analysis." *In re Soybean Futures Litig.,* 892 F.Supp. 1025, 1044 (N.D.Ill.1995). The test for manipulation "must largely be a practical one.... The aim must be therefore to discover whether conduct has been intentionally engaged in which has resulted in a price which does not reflect basic forces of supply and demand." *Cargill,* 452 F.2d at 1163.

***5** A number of manipulation cases under the CEA involve fraud, deceit, misrepresentation, or some violation of exchange rules by the defendant. *See, e.g., United States v. Reliant Energy Servs., Inc.,* 420 F.Supp.2d 1043, 1058 (N.D.Cal.2006). Those cases, however, do not support a conclusion that a plaintiff **must** show a defendant engaged in fraudulent activity or violated applicable trading rules to establish manipulation. Other cases addressing manipulation under the CEA demonstrate that a plaintiff need not show fraudulent conduct, a misrepresentation, or some violation of trading rules to establish manipulation. *See, e.g., In re Amaranth,* 587 F.Supp.2d at 535 ("[T]he combination of a wrongful intent (or more accurately, the lack of a legitimate economic motive) and a legitimate transaction would constitute manipulation."); *In re Henner,* 30 Agric. Dec. 1151, 1198 (U.S.D .A.1971).

The Court finds that to establish that an artificial price existed for the purposes of a CEA manipulation claim, a plaintiff need not establish fraud, misrepresentation, or a violation of exchange rules on the part of the defendant. *See Reliant,* 420 F.Supp.2d at 1058–59. While "fraud and deceit are not legitimate market forces," *see id.* at 1058, "[t]he methods and techniques of manipulation are limited only by the ingenuity of man." *Cargill,* 452 F.2d at 1163. *Cf. General Foods Corp. v. Brannan,* 170 F.2d 220, 224 (7th Cir.1948) ("[T]he common criteria **usual** in manipulation or corner cases are deceit, trickery through spreading of false rumors, concealment of position, the violation of express anti-manipulation controls, or other forms of fraud." (emphasis added)). Thus, the appropriate inquiry is whether the specific facts of a case to support a finding that the commodity price was determined by forces other than legitimate forces of supply and demand and whether a defendant intended to cause that artificial price.

Federal courts and the CFTC have provided useful guidance in how to determine whether the facts of a case support a CEA manipulation claim. *Cf. In re Ind. Farm Bureau,* Comm. Fut. L. Rep. (CCH) ¶ 21,796 at 27,281 (stating that the task of defining manipulation or attempted manipulation "has fallen to case-by-case judicial development"). For example, in *In re Amaranth,* the district court rejected the defendants' contention that a legitimate transaction, regardless of the intent of the defendants, cannot violate the CEA. *See In re Amaranth,* 587 F.Supp.2d at 533–34. The court held: "Because every transaction signals that the buyer and seller have legitimate economic motives for the transaction, if either party lacks that motivation, the signal is inaccurate. Thus, a **legitimate transaction combined with an improper motive** is commodities manipulation." *Id.* at 534 (emphasis added). The Court emphasizes that such a formulation is not the only manner in which to determine, on a case-by-case basis, whether an artificial price exists or whether a defendant caused an artificial price. *In re Amaranth,* however, illustrates one way that a court may review the sufficiency of a CEA manipulation claim.

2. United States v. Radley

***6** The Court is not persuaded that DFA's cited cases support its proposed rule. In particular, DFA relies on *United States v. Radley,* in which the district court addressed the sufficiency of a criminal indictment that charged a defendant with, *inter alia,* manipulation under the CEA. 659 F.Supp.2d 803, 806–09 (S.D.Tex.2009). The district court dismissed all counts charging defendant with manipulation or attempted manipulation because the CEA statute was constitutionally vague and, "[a]lthough the government has alleged that defendants caused and intended to cause an increase in price, it has not adequately alleged that the increased price was artificial." *Id.* at 816. The district court reasoned:

> Since defendants have not been accused of making false or misleading statements, the effect of their actions on the market was part of the legitimate forces of supply and demand.... Acting in a manner that shifts the price of a commodity in a favorable direction is the business of profitmaking enterprises, and if it is done without fraud or

> misrepresentation, it does not clearly violate the CEA.

Id. at 816. The district court further noted that "there is no universally accepted measure or test of price artificiality," and "[t]his uncertainty is the very thing that the constitutional vagueness doctrine is meant to protect against." *Id.*

Radley is not persuasive in the Court's analysis for two reasons. First, *Radley* is a criminal case and the district court in *Radley* did not attempt to define or limit what constitutes manipulation under the CEA, as suggested by DFA. Rather, *Radley* determined that Section 9 of the CEA was void for vagueness as a criminal statute because "a person of ordinary intelligence would not be able to determine that [the facts of the case as alleged in a criminal indictment] constitute price manipulation under the CEA." *Id.* at 812–13. *Radley's* analysis relating to the constitutional requirement of definiteness in a criminal statute is not helpful to the Court in these circumstances.

Second, *Radley's* and DFA's focus on a party's self-interested or profit-making motives misses the mark. (*See* Def.'s Mem. in Supp. of Mot. for Summ. J. at 11, Docket No. 103. ("[B]uying more of a commodity than needed to fill customer orders, with the intent of raising transaction prices, does not remove the purchases from the legitimate supply and demand for the commodity.").) Certainly, self-interest and profit-making motives do not remove a parties' conduct from the realm of legitimate forces of supply and demand. *See, e.g., In re Ind. Farm Bureau,* Comm. Fut. L. Rep. (CCH) ¶ 21,796. Classifying a motive as self-interested or profit-making, however, also does not render a party's conduct appropriate.

The Court finds it uncontroversial that an entity may act in its own self-interest or act with the intent to make a profit while trading in commodities and not run afoul of applicable trading rules or statutes. On the other hand, entities that act fraudulently or disseminate false reports also, presumably, are acting in their self-interest and for the purpose of making a profit, although fraud and deceit are not legitimate forces of supply and demand. In other words, generalizing an entity's motive as self-interested or profit-making does not remove the entity's conduct from the ambit of the CEA. Instead, the inquiry must be whether the facts of a case support a finding that defendant specifically intended to subvert legitimate forces of supply and demand.

***7** To prove that DFA intended to cause artificial prices on the CME Cheese Spot Call and on the Class III milk futures market, "it must be proven that [DFA] acted ... with the purpose or conscious object of causing or effecting a price or price trend in the market that did not reflect the legitimate forces of supply and demand." *In re Ind. Farm Bureau,* Comm. Fut. L. Rep. (CCH) ¶ 21,796 at 27,283. "Since proof of intent will most often be circumstantial in nature, manipulative intent must normally be shown inferentially from the conduct of the accused." *Id.*

The CME Cheese Spot Call is a thinly traded market, which during the relevant time period opened for only fifteen minutes a day and comprised only 2% of the sales of cheese in the United States. Yet, the price at which cheese traded on the CME Cheese Spot Call effectively set the price for cheese purchases nationwide. From May 21 to June 23, 2004, DFA purchased 323 loads of cheddar cheese blocks at $1.80 per pound. DFA was the sole purchaser of cheese on the CME Spot Call during that period. Plaintiffs allege that DFA's cheese purchases prevented cheese prices from dipping below $1.80 and that DFA was able to liquidate its long Class III milk futures contracts at a profit during the relevant time period. In addition, the price of cheese plummeted after DFA ceased purchasing cheese, indicating that DFA's purchases of cheese—allegedly without commercial need for the cheese—prevented the "determination of [cheese] prices by free competition alone." *See Socony–Vacuum Oil Co.,* 310 U.S. 150, 223 (1940); *see also Enron,* 2004 WL 594752, at *6 ("The CFTC alleges in its complaint that '[o]n July 19, 2001, artificial prices existed in the HH Spot Market, and in the NYMEX Henry Hub Futures as well.' The CFTC describes the time, market, and circumstances surrounding the 'price artificiality,' alleging that Shively engaged in a fifteen minute 'buying spree,' raising prices, and eventually unwound Enron's position in the market with a resultant price decline. The CFTC also points out that this activity caused prices in the NYMEX Henry Hub Futures Market to become artificial. The CFTC has alleged enough to survive a motion to dismiss." (citations omitted)). Viewing the facts in a light most favorable to plaintiffs, a reasonable trier of fact could find in plaintiffs' favor on their CEA manipulation claim.

Of course, the Court does not conclude as a matter of law that DFA's purchases of cheese on the CME Cheese Spot Call caused artificial prices for cheese or Class III milk futures or that DFA intended to cause an artificial price for cheese or Class III milk futures. Rather, the Court concludes only that based on the facts pleaded by plaintiffs and on the record,

there is a genuine dispute of fact as to whether the price of cheese and Class III milk reflected factors other than legitimate market forces of supply and demand and whether DFA intended to cause artificial prices. *See In re Soybean Futures,* 892 F.Supp. at 1058 (stating that "[a]s a general matter, ... questions of intent are inappropriate for resolution on summary judgment" but in some circumstances, dismissal may be appropriate if "the plaintiff presents no indication of motive and intent supportive of his position").

3. Legitimate Supply and Demand

***8** DFA argues that its purchases of cheese were part of legitimate supply and demand as a matter of law, and as a result, DFA asserts that no artificial prices for cheese or class III milk existed. In particular, DFA argues that federal courts and the CFTC have "held that market participants may legally purchase more of a commodity than needed to fill customer orders or at apparently higher than necessary prices." (Def.'s Mem. in Supp. of Mot. for Summ. J. at 10, Docket No. 103.)

The CFTC, however, has held that "[w]henever a buyer on the Exchange intentionally pays more than he has to **for the purpose of causing the quoted price to be higher than it would otherwise have been** ..., the resultant price is an artificial price not determined by the free forces of supply and demand on the exchange." *In re Henner,* 30 Agric. Dec. at 1198 (emphasis added); *accord Enron,* 2004 WL 594752, at *6. Plaintiffs have alleged and adduced evidence that DFA purchased cheese on the Cheese Spot Call to prop up Class III milk futures prices to enable DFA to liquidate its long Class III milk futures position at a profit, and plaintiffs allege that those actions did not constitute legitimate forces of supply and demand. *See In re Amaranth,* 587 F.Supp.2d at 534 ("[A] legitimate transaction combined with an improper motive is commodities manipulation."). DFA "treats as undisputed the factual allegations from Plaintiffs' Amended Complaint," (Def.'s Mem. in Supp. of Mot. for Summ. J. at 4 n. 4, Docket No. 103), and states that its motion is premised on a dispute over the appropriate legal standard, not on a dispute regarding the material facts, (Reply Mem. at 18, Docket No. 138). For the reasons discussed above, plaintiffs' factual allegations and the record show that there is a genuine dispute of fact as to plaintiffs' CEA manipulation claims, and summary judgment is not warranted.

III. MOTIONS TO EXCLUDE EXPERT OPINIONS

A. Standard of Review

Rule 702 of the Federal Rules of Evidence governs the admissibility of expert testimony. Fed. R. Evidence 702. Under Rule 702, proposed expert testimony must satisfy three prerequisites to be admitted. *See Lauzon v. Senco Prods., Inc.,* 270 F.3d 681, 686 (8th Cir.2001). First, evidence based on scientific, technical, or specialized knowledge must be useful to the finder of fact in deciding the ultimate issue of fact. *Id.* Second, the proposed witness must be qualified. *Id.* "Third, the proposed evidence must be reliable or trustworthy in an evidentiary sense, so that, if the finder of fact accepts it as true, it provides the assistance the finder of fact requires. *Id.* (internal quotation marks omitted). The district court has a "gatekeeping" obligation to make certain that all testimony admitted under Rule 702 satisfies these prerequisites. *Daubert v. Merrell Dow Pharm., Inc.,* 509 U .S. 579, 597–98 (1993). But an expert's opinion should be excluded as unreliable under the third prong only if that "opinion is so fundamentally unsupported that it can offer no assistance to the jury." *Bonner v. ISP Techs., Inc.,* 259 F.3d 924, 929–30 (8th Cir.2001) (internal quotation marks omitted).

B. Plaintiffs' Motion to Exclude DFA Expert James Jordan's Opinion on Mitigation of Damages

***9** Plaintiffs ask the Court to preclude DFA's lost-profits expert, James V. Jordan, from offering at trial his opinion relating to mitigation of damages. (Mot. to Exclude Test. of James V. Jordan, Docket No. 111; Mem. in Supp. of Mot. to Exclude at 1, Docket No. 112.) Plaintiffs argue that Jordan's expert opinion is contrary to law on the duty to mitigate and that Jordan's opinion is based on his "personal opinion." (Mem. in Supp. of Mot. to Exclude at 1, Docket No. 112.)

Plaintiffs assert that they incurred the majority of their damages on June 8 and June 9, 2004, when Class III milk futures reached levels that produced margin calls plaintiffs could not meet. Plaintiffs contend that as a result of DFA's manipulative scheme, which supported Class III milk futures prices, they suffered $4,343,613 in out-of-pocket losses. Similarly, Jordan estimated plaintiffs' "no mitigation" losses to be $4,339,436. (Jordan Expert Report ¶ 21, Garrod Decl. Ex. 1, Docket No. 120.) Jordan opines, however, that plaintiffs had a duty to mitigate, that plaintiffs failed to do so and, as a consequence, if plaintiffs succeed on the merits of their manipulation claim, plaintiffs may only claim less than $300,000 in damages. (*Id.* ¶ 31.) Plaintiffs seek to preclude Jordan from testifying that plaintiffs had a duty to mitigate damages prior to June 3, 2004, although plaintiffs concede

that Jordan may testify about his opinion on lost profits. (Mem. in Supp. of Mot. to Exclude at 4, Docket No. 112.) For the reasons discussed below, the Court grants plaintiffs' motion as to Jordan's opinion on mitigation of damages as contrary to law.

1. Jordan's Expert Opinion

DFA retained Jordan as its lost-profits and damages expert. Jordan's expert opinion and testimony state that plaintiffs' "gross out-of-pocket losses of over $4 million were the result of large, risky short positions in milk futures, which [plaintiffs] maintained in the face of increasing price volatility (i.e., increasing risk), and persistent indications that [their] market view was wrong." (Jordan Expert Report ¶ 22, Garrod Decl. Ex. 1, Docket No. 120.) Jordan opines that "regardless of the alleged market effects of DFA, ... [plaintiffs] should have mitigated [their] losses by reducing or eliminating the [short] positions as market volatility increased and as market prices continually moved contrary to [their] market view." (*Id.*)

Jordan concludes that plaintiffs accumulated significant losses between May 28 (when they could have exited the market with $500,000 in profit) and June 9 (when plaintiffs were forced to liquidate their Class III milk futures at a loss of millions of dollars). (*Id.* at 8 & Ex. 1B.) Jordan concludes that given the market information of which plaintiffs were aware, "exiting the market by approximately June 3 is a reasonable mitigation assumption," and Jordan therefore calculated plaintiffs' gross out-of-pocket damages at no more than $501,327. (*Id.* ¶ 31.)

***10** Jordan notes that between May 14 and May 25, plaintiffs increased their short Class III milk futures position from 631 contracts to the maximum of 1575 contracts. (*Id.* ¶ 23.) Jordan states that plaintiffs' short position as of May 25 "exposed [them] to a substantial risk of loss." (*Id.* ¶ 24.) Jordan notes that plaintiffs reduced their short position to 1100 contracts through June 3 and to 900 contracts on June 8, before margin calls forced plaintiffs to liquidate their short Class III milk futures contracts. (*Id.* ¶ 25.) Jordan concludes that "[t]he risk of [plaintiffs'] large short positions would have been obvious to [plaintiffs] from the large profit/loss swings [they] was experiencing during [the relevant] period." (*Id.* ¶ 26.)

Jordan states that there were two circumstances that should have contributed to plaintiffs' awareness of the risks inherent in holding large short positions in Class III Milk futures contracts. First, Jordan contends that there was significant milk market volatility in May and June that could be seen by simply observing the market. (*Id.* ¶¶ 25–27.) Second, Jordan concludes that there were "signs" making it "[e]qually obvious to [plaintiffs] by May 25 ... that market prices were beginning to behave contrary to [their] market view." (*Id.* ¶ 28.) Jordan contends that prior to June 4, "[plaintiffs] could have recognized the riskiness of [their] large short positions and the increased market volatility and elected to reduce or eliminate [their] exposure to the risk of large losses.... [Plaintiffs] took undue risk by not reducing or eliminating his exposure to losses in early June." (*Id.* ¶ 29.) In short, Jordan opines that the market signals indicating to plaintiffs that they "had been wrong in [his] assessment of the market," were the same signals that plaintiffs saw from May 21, 2004, to June 9, 2004, when plaintiffs finally liquidated their short positions. (*Id.* ¶ 30.)

At his deposition, Jordan elaborated on the background principles he applied in reaching his expert opinion. As to his understanding of the "concept of mitigation," Jordan stated, "if a person or institution is at—is in a position where they could be harmed, ... they should take reasonable steps to prevent that harm, prevent or reduce their harm, regardless of the cause of their harm." (Jordan Dep. Tr. 125:23—126:4, Oct. 22, 2009, Garrod Decl. Ex. 2, Docket No. 120.) Jordan did not testify that he thought plaintiffs should have known that DFA was manipulating the market at that time, but instead confirmed "that [plaintiffs] had a duty to avoid harm before [they] even knew that the market was being manipulated." (*Id.* 126:5–16.) In reaching his opinion, Jordan testified:

> I'm applying a reasonableness standard, and it is based upon whether the individual had information that would have allowed him to conclude that he was in a highly risky position and could readily sustain very large losses in the future.
>
> ***11** And so at that point, the individual has sufficient information to draw a conclusion that I'm really exposed here, this is extremely risky, and that that meets the standards of mitigation analysis, I believe, that you—you ask yourself at what point would reasonable behavior by the individual indicate mitigation.

(*Id.* 131:5–16.) Jordan testified that June 3, 2004, was "a reasonable date at which [plaintiffs] had enough information to have taken reasonable steps to reduce the potential for loss." (*Id.* at 127:1–4.)

2. Contrary to Law

Jordan's mitigation opinion is contrary to law and therefore would not be helpful to a finder of fact in deciding the issues in the case. "As a general rule, a party defrauded cannot, **after discovery of the fraud,** increase his damages by continuing to expend money on the property retained and recover for such expenditures." *Clements Auto Co. v. The Serv. Bureau Corp.,* 298 F.Supp. 115, 136 (D.Minn.1969) (emphasis added; internal quotation marks omitted). The duty to mitigate, however, does not arise before an individual has knowledge of the wrongdoing. *See, e.g., Connelly v. Hyundai Motor Co.,* 351 F.3d 535, 542 (1st Cir.2003) ("[T]here is no duty to mitigate damages **prior to sustaining an injury[.]**" (emphasis added)); *Nilson–Newey & Co. v. Ballou,* 839 F.2d 1171, 1175 (6th Cir.1988) (holding that the duty to mitigate damages "arises only after the defendant's tortious conduct, not before it"); *Arrington v. Merrill Lynch, Pierce, Fenner & Smith, Inc.,* 651 F.2d 615, 620 (9th Cir.1981) ("Plaintiffs' damages in a 10b–5 case are limited by what they would have realized had they acted to preserve their assets or rights when they first learned of the fraud or had reason to know of it."); *Harris v. Am. Inv. Co.,* 378 F.Supp. 894, 900 (E.D.Mo.1974) ("[A] plaintiff has a duty to mitigate his damages **upon discovery of the fraud.**" (emphasis added)).

The parties agree that "[commodity] future customers have a general obligation to mitigate damages." *Samson Refining Co. v. Drexel Burnham Lambert, Inc.,* No. 82–R448, Comm. Fut. L. Rep. (CCH) ¶ 24,596, 1990 CFTC LEXIS 90 (Feb. 16, 1990). However, "complainant's duty to complain about unauthorized trading **does not arise until it learns of the wrongdoing.**" *Id.* at 117 (emphasis added). "[A] customer should not be allowed to recover damages for losses occurring after the customer learns of the truth, can reasonably foresee that further damages are likely and does not act reasonably to limit his damages." *Id.* at 115–16 (internal quotation marks omitted); *see also Darrah v. First Am. Inv. Servs. Co.,* No. 05–R042, 2006 CFTC LEXIS 59, at 103 n. 23 (June 28, 2006) ("The duty to mitigate doesn't arise until complainant becomes aware of the underlying wrongdoing.").

Jordan states: "[plaintiffs] had a duty to avoid harm before [they] even knew that the market was being manipulated." (Jordan Dep. Tr. 126:5–16, Oct. 22, 2009, Garrod Decl. Ex. 2, Docket No. 120 .) In effect, Jordan opines that a commodities trader must take constant precautions against fraud in light of the fact that someone, somewhere, may be perpetrating a fraudulent trading scheme. Such a standard is not supported in the law.

***12** Further, a standard that requires an individual to mitigate damages when he "should have known" of the wrongdoing—as proposed by DFA—is impractical. As the Seventh Circuit stated:

> The best solution is for people not to harm others intentionally, not for potential victims to take elaborate precautions against such depradations. If the victims' failure to take precautions were a defense, they would incur costs to take more precautions (and these costs are a form of loss victims would feel in every case, even if the tort does not occur), while would[-]be tortfeasors would commit additional torts because they would not fear the need to pay up in cases where the victims do not protect themselves. Common law torts have balked at such an outcome in ordinary tort cases, and securities law has followed the same path.

DeRance, Inc. v. PaineWebber Inc., 872 F.2d 1312, 1323 (7th Cir.1989). Thus, proof that a victim "should have known" of the wrongdoing does not trigger a duty to mitigate, although if the victim **knew** of the wrongdoing and did nothing or "consciously disregarded information" that would have informed him of the wrongdoing, he may not complain. *Id.* at 1324.

In sum, Jordan's mitigation opinion is directly contrary to the law, and the opinion is therefore not helpful to a fact-finder at trial. The Court thus precludes Jordan from testifying at trial regarding Anderson's duty to mitigate.

C. Anderson's Motion to Exclude Expert Testimony of Robert Mackay and DFA's Motion to Exclude Expert Testimony of Wayne Brown

Anderson moves to exclude certain portions of DFA expert Robert McKay's opinion relating to (1) the legal definition of manipulation; (2) causation; and (3) intent under the Commodities Exchange Act. (Mot. to Exclude Test. of Robert J. McKay at 1, Docket No. 127.) DFA retained McKay to provide an expert opinion on whether DFA caused artificial

prices on the Spot Cheese and Class III milk futures markets and to refute the expert opinion of Anderson's expert, Wayne Brown. (MacKay Expert Op. ¶ 13, Garrod Decl. Ex. 1, Docket No. 129.) MacKay concludes, *inter alia,* that DFA did not manipulate either the CME spot cheese market or the Class III milk futures market between May 21, 2004, to June 23, 2004. (*Id.*)

DFA moves to preclude plaintiffs' expert, Wayne Brown, from offering at trial expert testimony regarding whether DFA manipulated the CME Cheese Spot Call and Class III milk futures markets and regarding plaintiffs' damages. (Mem. in Supp. of Mot. to Exclude Ops. of Wayne R. Brown at 1, Docket No. 117.) Brown offers two opinions. First, Brown concludes "that DFA manipulated the price of cheese and milk futures when it defended the price of cheese as quoted on the CME from May 21 through June 23, 2004 with cheese purchases in order to unwind its investment in milk futures." (Brown Expert Report ¶ 12, Mansfield Decl. Ex. 1, Docket No. 118.) Brown opines that "[b]ecause of the relationship between cheese and milk futures, DFA manipulated the price of milk futures." (*Id.*) Brown states that "DFA had 1) the ability to influence prices; 2) the specific intent to create an artificial price; 3) the artificial price existed[;] and 4) causation existed." (*Id.*) Second, Brown concludes that given plaintiffs' out-of-pocket losses and lost profits—which were the result of DFA's manipulative activities on the CME Cheese Spot Call—"the most reasonable damages amount is $11,769,253." (*Id.* ¶ 13.)

***13** Plaintiffs and DFA prematurely brought their respective motions to exclude. Both parties filed their motions prior to the Court's ruling on DFA's motion for summary judgment, which addresses the appropriate standard for reviewing a CEA manipulation claim. As a consequence, the parties largely repeat the arguments made at summary judgment. The Court therefore dismisses without prejudice plaintiffs' motion to exclude the expert testimony of Robert Mackay and DFA's motion to exclude the expert testimony of Wayne Brown. The parties may, prior to trial, submit motions to exclude portions of those experts' testimony in light of the Court's ruling on the motion for summary judgment.

This case will be placed on the Court's next available trial calendar.

ORDER

Based on the foregoing, and all the files, records, and proceedings herein, **IT IS HEREBY ORDERED** that:

1. Defendant Dairy Farmers of America, Inc.'s Motion for Summary Judgment [Docket No. 102] is **DENIED.**

2. Plaintiffs Mark Anderson and Killer Whale Holdings, LLC's Motion to Exclude the Testimony of James V. Jordan [Docket No. 110] is **GRANTED.**

3. Defendant Dairy Farmers of America, Inc.'s Motion to Exclude Opinions of Wayne R. Brown [Docket No. 115] is **DENIED without prejudice.**

4. Plaintiffs Mark Anderson and Killer Whale Holdings, LLC's Motion to Exclude the Testimony of Robert J. Mackay [Docket No. 127] is **DENIED without prejudice.**

Footnotes

1 For the purposes of the motion for summary judgment, DFA states that it treats as undisputed Anderson's factual allegations pleaded in the Amended Complaint. (Def.'s Mem. in Supp. of Mot. for Summ. J. at 4 n. 4, Docket No. 103.)

2 DFA prefaced this statement to the CFTC by stating that it was assuming certain facts for the purposes of the letter, but "[b]ecause the Division and DFA are continuing to gather and confirm certain information ... the Division should not consider the following assumed facts to be factual assertions by DFA. Furthermore, if additional or different material facts come to light, DFA will revise its analysis if necessary." (Garrod Decl. Ex. 1 at 2, Docket No. 124.)

3 DFA argues that its purchases of cheese were a part of legitimate forces of supply and demand, and that an "intent to influence price" does not equate to an "intent to cause an artificial price." In the Court's view, DFA's arguments address a single issue relating to Anderson's establishment of a CEA manipulation claim: whether plaintiffs must show that DFA committed fraud, engaged in sham or fictitious transactions or cornering activity, traded in a manner that disrupted orderly trading processes, or violated applicable trading rules. Accordingly, although the two elements are distinct and must be separately proven, in these circumstances, the Court addresses the question of whether artificial prices existed together with the element of "intent to cause an artificial price." *Cf. In re Soybean*

Futures Litig., 892 F.Supp. 1025, 1057 (N.D.Ill.1995) ("[T]here is no universally accepted measure or test of price artificiality, and ... this element can be closely interrelated with the other three elements of a manipulation claim[.]").

EXHIBIT 11

2012 WL 2874059
Only the Westlaw citation is currently available.
United States District Court,
C.D. Illinois.

Tanya NUNEZ, Administrator of Estate of Cynthia L. Madden, deceased, Plaintiff
v.
BNSF Railway Company, Defendant.

No. 09–4037. | July 13, 2012.

Attorneys and Law Firms

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Opinion

ORDER

JOHN A. GORMAN, United States Magistrate Judge.

***1** The parties have consented to have this case heard to judgment by a United States Magistrate Judge pursuant to 28 U.S.C. § 636(c), and the District Judge has referred the case to me. Now before the court are the Defendant's motions to bar Plaintiff's two expert witnesses, James Sottile (# 58) and Paul Bodnar (# 56). The motions are fully briefed. As explained below, both motions are **GRANTED.**

BACKGROUND

On the night of May 28, 2007, Cynthia Madden appeared to be having car trouble. Her car stalled several times, the last time at the point on Cleveland Road where it intersects with railroad tracks in Colona, Illinois. As Ms. Madden tried to re-start her car, two motorists in the area saw the crossing gates came down and the warning lights begin to flash. One of them heard a train horn blowing. Both of them observed that Ms. Madden did not immediately get out of her car. It was not until the train was visible to those at the crossing that Ms. Madden fled her car. She was too late. The train hit her car, which in turn struck her. She did not survive.

The train was a BNSF train. The engineer and conductor on board both recall that the train's headlights were on and that horn was sounded for more than 20 seconds before it entered the intersection. Both stated that the train, as it approached the intersection, was traveling under the allowable speed of 30 m.p.h. The conductor recalls seeing the closed gates and flashing lights at the intersection. They agree that, as Ms. Madden's car came into view, the train was immediately thrown into emergency mode, but it was not possible to stop the train in time.

The Cleveland Road crossing is protected by what is referred to as an "active warning system," meaning that as a train approaches the crossing, warning lights start flashing, gates automatically lower, and pedestrian bells ring. This system is triggered by circuits placed in the tracks that sense a train's approach. When a train is sensed, the system performs a rapid calculation, based on the train's speed and the distance to the intersection, that triggers the active warning system.

This warning system produces data (such as the train's speed and activation of the active warning system) that is transmitted to an event recorder. [1] There are two parts to this event recorder: the HXP–3, which operates the warning system and produces the data, and the HCR, which records the data and allows it to be downloaded to a computer and printed. When the event recorder is installed, there are various "options" that must be selected. In addition, the installer may select "daylight savings time" as an option. In the system at the Cleveland Road crossing, that option had not been selected, meaning that the recorded data did not reflect daylight savings time.

There is a second event recorder as well, this one found in the locomotive itself. This event recorder continuously records data specific to operation of the locomotive, including such data as speed, direction, and time. The specific locomotive on this train was BNSF 4708, which was manufactured in 1997.

***2** About 2 hours after the accident, BNSF personnel downloaded the event recorder at the crossing. The morning after the accident, BNSF personnel downloaded the locomotive's event recorder. Plaintiff was provided with a copy of these downloads. In addition, BNSF tested the lights, horn and brakes on the locomotive. The original test results were no longer available by the time this litigation began. Federal regulations only require that the test results themselves be kept until the next test is conducted, but "in no case for less than one year from the date of the test." 49

C.F.R. § 234.273. The Plaintiff was provided with a summary of the tests that BNSF conducted. This summary showed that no problems were found on the test conducted immediately after the accident.

Several days after the accident, Ms. Madden's children went to the scene to retrieve her personal belongings. While they were there, they heard a train horn and, using a cell phone and two wrist watches, they timed the arrival of the train at the crossing. One of the children testified that the arrival of the train at the crossing was 13–14 seconds and that the "lights came on two or three seconds before the train went through."

It was not until October 28, 2011, that Plaintiff retained 2 expert witnesses, Paul Bodnar and James Sottile. One or both of these experts, along with counsel for the parties and a BNSF representative, visited the Cleveland Road crossing site in November. In addition to viewing the physical layout of the scene of the accident, BNSF opened the "bungalow" that contains the HCR portion of the event recorder for the tracks. Contained within that "bungalow" was a document reflecting the most recent 2011 test of the warning system. The expert obtained a photograph of that document.

The two experts authored a joint report containing five opinions. Paul Bodnar's sole opinion is that BNSF was negligent because the train did not sound its horn prior to entering the intersection. James Sottile expresses four opinions: (1) that the times on the data downloaded from the recorder are inaccurate; (2) that the active warning system gave only 13–14 seconds of warning, less than the 20 seconds required by 40 CFR § 234.225; (3) that the crossing signal system appliance test records provided by BNSF do not meet federal requirements [49 C.F.R. 234.273]; and (4) because the signal event recorder data fails to comply with federal regulations, the records are insufficient to determine the speed, horn and active warning systems at the time of the accident. Their joint Report was served on the Defendant, which has now moved to bar their testimony in full. A hearing on the motions was held on June 12, 2012.

PROCEDURAL HISTORY

In order to fully comprehend the motions attacking the expert's Report, it is necessary to review the procedural and discovery history in this case. This lawsuit was filed on May 22, 2009. The Rule 16 scheduling conference was held on September 23, 2009. The parties' plan was approved. That plan called for Plaintiff to disclose expert witnesses by January 15, 2011, for Defendant to disclose expert witnesses by April 16, 2011, and for all discovery, fact and expert, to close on May 20, 2011. The Court was not involved in the case again until the Plaintiff moved for an extension of the schedule. Following a conference on February 17, 2011, the schedule was extended. The new schedule closed all discovery—fact and expert—on December 16, 2011.

***3** On November 9, 2011, Plaintiff made the request (or, as Defendant characterized it, the demand) that Plaintiff's expert be allowed to conduct an inspection of the accident scene. Defense counsel agreed, but only with Plaintiff's counsel's representation that the expert's report would be provided before Thanksgiving and the deposition taken before Christmas.

On November 17, 2011, a month before the conclusion of all discovery, the chambers of the undersigned was contacted by counsel for BNSF, who orally requested a hearing. At that time, the parties' attorneys were present at the scene with Plaintiff's expert and a representative from BNSF. The expert had asked BNSF to open the "bungalow" in which the HCR event recorder is stored, a request to which BNSF objected as beyond what had been agreed to. A hearing was held, and the Court directed that the bungalow be opened for inspection and photography by the expert.

At 6:43 p.m. on December 16, 2011, the final day of discovery, Plaintiff served her experts' Report. On December 19, 2011, Defendant filed a motion to bar Plaintiff's experts on the grounds of untimeliness. On December 21, Defendant filed a supplement to that motion, stating that the expert had served a "revised" report on December 19 at 8:58 p.m. In response, Plaintiff attempted to justify its late production of the expert's report by pointing to what she characterized as BNSF's own delays in producing documents, its refusals to produce documents, and deficiencies in the documents that BNSF had produced. For good measure, Plaintiff included substantive arguments about the merits of the case.

In an Order entered on January 24, 2012, the Court entirely rejected Plaintiff's arguments, pointing out first that any disputes about the sufficiency of document production should have been brought to the Court's attention during discovery; by waiting until after discovery closed, any problems were waived. The Court found "puzzling" Plaintiff's lack of diligence in alerting the Court to any such deficiencies, because Plaintiff's explanation for taking no depositions of

BNSF personnel during the course of discovery was her counsel's belief that the case could be proved with only documents and experts.

After serious consideration being given to barring the experts for failure to timely disclose them and their Report, the Court decided that, in the interests of justice, the motion to bar should be denied. Instead, a new schedule was implemented, which allowed BNSF 7 days to review its production of documents to ensure that it had been complete, and allowed Plaintiff's experts 7 days thereafter to revise their Report if BNSF produced any additional documents. The Order cautioned, in bold, capital letters, that "NO EXTENSIONS OF THESE DEADLINES will be allowed without a detailed and substantial showing of good faith and due diligence."

BNSF produced no additional documents. Despite the strong cautionary language of the Order, on February 2 Plaintiff filed a motion to compel discovery. In that letter, counsel stated that he had been unavailable to work on this case until after January 30[2]. On January 31, he reviewed the discovery he had previously requested and served a supplemental request for what he believed was missing. BNSF counsel responded that no additional documents would be provided. Hence, the motion to compel.

***4** As was pointed out in Defendant's response to this motion, Plaintiff's argument was based on two misconceptions. The first was that a defendant must, in its Rule 26(a) disclosures and supplements, provide documents that support the *plaintiff's* claim. That is not what the Rule requires. It requires a party to disclose information that will support "its" claims or defenses. Then, the opposing party uses those disclosures to formulate its subsequent written and oral discovery.

The other misconception was that the Court's January 24 Order required BNSF to forgo any objections it had previously made and simply produce everything that Plaintiff had requested. The Order required BNSF to produce documents it "should have" produced during discovery. If objections were made and Plaintiff did not timely file motions with respect to those objections, then BNSF was not obligated to withdraw those objections to be in compliance with the Order. Plaintiff's motion was denied.

The January Order also set February 24 as the deadline for Defendant to depose Plaintiff's experts. BNSF took the depositions of the Plaintiff's experts in a timely fashion. Defendant's deadline to disclose its expert was March 9, 2012. The expert and his report were timely produced, but Plaintiff did not take his deposition.

EXPERT WITNESSES GENERALLY

The admissibility of expert testimony is governed by Federal Rules of Evidence 702 and 703, as well as the Supreme Court's opinion in *Daubert v. Merrell Dow Pharm., Inc.,* 509 U.S. 579 (1993); *Lewis v. CITGO Petroleum Corp.,* 561 F.3d 698, 705 (7th Cir.2009).

Expert opinion testimony is admissible, so long as it conforms to Fed.R.Evid.702, which provides:

> A witness who is qualified as an expert by knowledge, skill, experience, training or education may testify in the form of an opinion or otherwise, if:
>
> (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
>
> (b) the testimony is based on sufficient facts or data;
>
> (c) the testimony is the product of reliable principles and methods; and
>
> (d) the expert has reliably applied the principles and methods to the facts of the case.

Under Fed.R.Evid. 703, an expert may base his opinion on facts that are in the record or on facts that are presented to him or on facts that he personally observes. Under this Rule, expert testimony must be rejected if it lacks an adequate basis in fact. *Cella v. U.S.,* 998 F.2d 418 (7th Cir.1993). Evaluation of the "soundness of the factual underpinnings" are crucial to the Court's gatekeeping function. *Smith v. Ford Motor Co.,* 215 F.3d 713, 718 (7th Cir.2000). An expert may not simply ignore evidence that does not support his opinion. See, e.g., *Barber v. United Airlines Inc.,* 17 Fed. Appx.433 (7th Cir.2001).

Under *Daubert,* the district court acts as a gatekeeper to ensure that expert testimony is both relevant and sufficiently reliable. *Kumho Tire Co. v. Carmichael,* 526 U.S. 137, 147 (1999)); see also *Mihailovich v. Laatsch,* 359 F.3d 892, 918 (7th Cir.2004) and *Bielskis v. Louisville Ladder,* Inc., 663 F.3d 887, 893 (7th Cir.2011).

***5** Whether to admit expert testimony rests within the discretion of the district court. See, e.g., *Gen. Elec. Co. v. Joiner,* 522 U.S. 136,(1997). Indeed, a district court has "wide latitude in performing its gate keeping function and determining both how to measure the reliability of expert testimony and whether the testimony itself is reliable." *Bielskis,* 663 F.3d at 894. The inquiry under Rule 702 is "flexible." *Id.* "The goal of *Daubert* is to assure that experts employ the same 'intellectual rigor' in their courtroom testimony as would be employed by an expert in the relevant field." *Jenkins v. Bartlett,* 487 F.3d 482, 489 (7th Cir.2007) (quoting *Kumho Tire v. Carmichael,* 526 U .S. 137, 152 (1999) *Kumho Tire Co.,* 526 U.S. at 152). The court's role is that of "gatekeeper" with respect to expert testimony. *Kumho Tire,* 526 U.S. at 147.

District courts employ a three-part analysis before admitting expert testimony: the expert must be found qualified to provide an opinion on the particular subject; the expert's methodology must be found reliable; and there must be a relevant connection between the methodology and the opinion proffered. *Daubert.* at 509 U.S. at 589–92. In other words, the expert must be qualified and the opinions must be both relevant and reliable. *Id.* at 589.

The opinion must assist the trier of fact is some material way. See, *Dhillon v. Crown Controls Corp.,* 269 F.3d 865, 871 (7th Cir.2001). Where an expert has "unjustifiably extrapolated from an accepted premise to an unfounded conclusion," the gap between the facts and the opinion is simply too great to be helpful or admissible. *G.E. v. Joiner,* 522 U.S. 136, 146 (1997).

Defendant does challenge the qualifications of Sotille and Bodnar to offer the expert opinion testimony at issue. For purposes of this Order, however, it is assumed that the two witnesses are qualified generally to testify about railroad safety and regulations. For the most part[3] Defendant's arguments as to the expert witness's qualifications either go to the substance or the weight of the opinions. The former is dealt with below; the latter is an issue for a trier of fact. The issue before the Court is the methodology used by and/or the substance of the opinions tendered by these witnesses.

BODNAR'S OPINION

Bodnar was tendered as an expert in train operating practices regarding the horn and in interpretation of the locomotive event recorder data. His specific opinions concern the horn on the locomotive. In his report, he draws 3 conclusions: (1) the BNSF employee who downloaded the locomotive event recorder did not request the proper parameters for the download, rendering the download incorrect, incomplete and impossible to interpret; (2) the locomotive horn was removed for testing; and (3) the locomotive horn did not sound for the required 15 seconds before it reached the intersection.

Bodnar's first opinion is based on the fact that the locomotive data download included no data showing activation of the horn, although the event recorder installed on this locomotive was capable of recording such data. Bodnar asserts that, because federal regulations require recording this information, the failure to record such data indicates four possibilities: (1) the entries were deleted; or (2) the horn was never activated by the engineer; or (3) the horn failed mechanically; or (4) the event recorder failed to record activation of the horn by the engineer.

***6** The regulation cited by Bodnar is inapplicable. This locomotive was manufactured in 1997. The regulation he cited applies to locomotives placed in service after October 1, 2009, a regulation that obviously has no applicability to a locomotive involved in an accident that occurred in 2007. The pertinent regulation does not require recording or retention of data relating to the horn on the particular locomotive involved in this case. 49 C.F.R. § 229.135. Bodnar's underlying assumption that the failure to record this data is a serious deficiency is without merit.

When Bodnar was confronted with the proper regulation during his deposition, he challenged whether it was a "current" regulation and said he would have to check with his partner to confirm that it was. Even if it was the current regulation, however, Bodnar opined that if BNSF had upgraded its recorder so that horn data *can* be recorded, it *should* be recorded. (Bodnar Deposition p. 67–8). This is contrary to law. See, *Waymire v. Norfolk and Western Ry. Co.,* 218 F.3d 773, 776 (7th Cir.2000)(railroad not liable in FELA negligence action for unsafe speed and inadequate warning devices if railroad's conduct was consistent with regulations).

In addition to the lack of legal support for the proposition that the horn data was deficient, Bodnar suggests four other possibilities (besides the lack of any obligation to record the data) that would explain why no horn data showed up.

None of these four possibilities is viable, given the undisputed factual evidence in the record.

Bodnar admitted in his deposition that the first possibility —entries for the horn had been deleted—was wholly unsupported by any evidence (p.94), and no such evidence has been brought to the Court's attention. In addition, by its very definition, an event recorder must be tamper proof, so deletion of data would not appear to be a possibility anyway. This possibility is nothing more than pure speculation.

Bodnar insisted at his deposition that the lack of recorded data is evidence to support his second possibility—that the horn was never activated at all. This flies in the face of undisputed testimony from three witnesses—one of them completely independent of BNSF. Bodnar discounts witness testimony as unreliable, but this is not a situation of conflicting witness testimony; there is no contrary witness testimony. As BNSF points out, Bodnar's opinion on this point is like saying that if a tree falls in the forest but there is no video recording of it, the tree never fell even if witnesses observed it. An expert may not simply ignore evidence that does not support his opinion.

The third possibility posited by Bodnar-that the horn failed mechanically—is also belied by the evidence. The data download and the test result summaries reviewed by Bodnar during his deposition all show that the horn was tested as a matter of routine before the accident and again after the incident. No deficiency in the horn was found at any time. Bodnar cited no evidence at all to support this as a viable possibility-other than the lack of horn data on the download, and Plaintiff cites none in the response to this motion.

***7** The fourth possibility stated by Bodnar was that the event recorder failed to record activation of the horn by the engineer. To the extent that this implies some malfunction in the event recorder, no evidence is cited in the Report or in Plaintiff's response to this motion that would support such an implication and it is contrary to the test records produced by BNSF. It could also be read as simply reflecting the reality that activation of the horn was not recorded because there was no requirement to do so. If read in that manner, the statement attributes no wrongful conduct to BNSF. Neither interpretation of this "possibility" is helpful in the least. It could be read as simply reflecting the reality that activation of the horn was not recorded because there was no requirement that it be recorded. If read in that manner, the statement attributes no wrongful conduct to BNSF. Neither interpretation of this "possibility" is helpful in the least.

Not one of the four possibilities posited by Bodar supports his opinion that the data download from the event recorder on the locomotive was deficient because it did not record activation of the horn. This opinion is not only contrary to evidence but also without factual support, and it is legally unsupportable. It is therefore not admissible.

Bodnar's second opinion is that, contrary to good practices in the industry, the horn in the locomotive had been removed for testing, suggesting some sort of cover-up. When challenged by BNSF to support that assertion, he acknowledged that the documents he was shown during his deposition showed that the horn was still mounted on the locomotive when the testing was conducted. He testified, however, that he thought he had seen some other document showing removal of the horn and that he would identify it after his deposition if he located it. (p.112–13). It is not disputed: no such document has been produced. This opinion is not based on any evidence and is therefore inadmissible.

Bodar's third opinion—that the locomotive horn did not sound for the required 15 seconds before it reached the intersection —is based on certain "calculations" that he performed once he became convinced that the train was traveling at 26 miles per hour. He calculated that 10 seconds elapsed from the time the locomotive was put into emergency mode (which was when the engineer saw Ms. Madden's car on the tracks) to the time of the collision. From that statement, he concludes that the horn was not sounded during those 10 seconds and it is not possible to tell if the horn was sounded during the preceding 5 seconds of the required 15 second interval during which the horn was required to sound. There is no explanation, either in his report or in his deposition, for how he reached the conclusion that the horn was silent during those final 10 seconds. To the extent this conclusion is based on the fact that the sounding of the horn was not recorded, it is without merit as discussed above. If there is some other basis for the conclusion, it has nowhere been stated and, as noted earlier, is contrary to witness testimony. The leap of logic required to reach the conclusion is simply too great. This opinion is inadmissible.

***8** One other problem is raised by Defendant, and that is the fact that Bodnar had his partner review his work, but Bodnar failed to disclose that fact until during his deposition. While this is a serious shortcoming—an expert must disclose all bases for his opinions, Fed.R.Civ.P. 26(a)(2)(B(i)-in this situation it proves relatively harmless, since Bodnar's

opinions are, in any event, inadmissible. Nonetheless, this shortcoming would in the ordinary course of litigation arguably be subject to Rule 37 sanctions. As noted above and as noted in previous orders, if Plaintiff intended to prove this case using only experts and documents, the failure to use care in timely and completely complying with the Rules is simply inexplicable.

Bodnar's opinion is not based on objective or scientific evidence but rather on Bodnar's purported knowledge of various safety rules that apply to railroads such as BNSF. His knowledge of the applicable rules demonstrated shortcomings, and he attempted to apply those rules without factual support and with disregard to objective facts. His opinion that "the horn wasn't sounded in time for Ms. Madden to have that window of opportunity to step away"-which encompasses each of the three separate opinions discussed above-fails to meet the most basic standards of the Rules of Evidence and *Daubert*. The motion to bar Bodnar from testifying is therefore granted.

SOTTILE'S OPINIONS

James Sottile's area of expertise is signals and train control. His portion of the Report offered 4 opinions relating to what the signal event recorder data download showed about the train's movement and the signal operations on the date in question.

His first "opinion" is that the wayside event recorder did not record in daylight savings time. This is not an opinion at all; it is an undisputed fact. BNSF does not assert that the recorder did record in daylight savings time.

Sottile's point in making this observation is that he cannot be certain of the timing of various events due to the failure to record in daylight savings time. He refused in his deposition to acknowledge that the time stamp on the signal event recorder downloaded data can simply be adjusted by one hour to obtain the correct time for the events recorded. He insisted that making this adjustment would be an "assumption," and he refused to make that assumption (p.124).

It is not an "assumption" to make this adjustment. If everyone agrees that the time stamps are in central time, not in central daylight time, then no assumption is required to determine the actual time that events occurred. It simply defies common sense to refuse to acknowledge this. His opinion on this matter is unreasonable and entirely unhelpful. It therefore fails to meet Rule 702's requirement that the opinion assist the jury; in fact, allowing this testimony would likely confuse the jury. It is, moreover, not relevant to any issue of negligence or causation. Because it is neither relevant nor helpful, this "opinion" is not admissible.

***9** Sottile's second opinion is that the active warning system gave only 13–14 seconds of warning, less than the 20 seconds required by 40 CFR 234.225. This opinion is not based on any tests conducted by Sottile or on any data provided by BNSF. It is instead based on timing tests conducted by the decedent's children.

The children's test was conducted using their cell phones' stop watch function and their wristwatches to time a different train on a different day with no knowledge of the train's speed. They began timing when they heard the train, to see how long it was before the train entered the intersection. Plaintiff argues that timing devices such as cell phones and watches are accurate devices and that this test was certainly valid information on which the expert could rely.

BNSF does not challenge the accuracy of the timing devices. It challenges instead the ability of a lay person to use these devices to record accurately a time that begins with the lay person's hearing the horn of the train. When to start and stop the device would be key. As BNSF points out, when something is "heard" is "a completely subjective and highly available point in time." Did they start timing when they heard the train engine? heard the whistle? or, as the active warning system operates, when the train reached a certain point on the track? Where was the train when they stopped timing? Sottile testified (p.128) that if he had been the accident investigator and had the information about the children's timing, he would have used that information to order further testing. He did not testify that he would have based any conclusive opinion on their timing.

Moreover, when he was challenged by the documentary evidence at his deposition, Sottile agreed [4] that there were 30 seconds of active warning time prior to the accident. (p.88–90). This does not just undercut the opinion he stated in the Report; it contradicts it.

Sottile also references that one of Madden's children recalled that someone said something at the coroner's inquest about the train's speed being 50–60 mph. There is no such evidence in the record. The Court assumes that if evidence of such

speed was presented at the inquest, Plaintiff would have presented it to the Court, and this case would be a very different case. As it is, Sottile testified in his deposition that the locomotive event recorder showed that the train was traveling at 28 mph when it was placed into emergency mode by the crew (p. 142–143). He acknowledged that the data from the train dispatcher showed that the speed of the train "in the neighborhood" of 30 mph. (p. 55). All the documentary and testimonial evidence shows that the train's speed was 30 m.p.h. or less. For Sottile to have relied in any way on this type of secondhand information under these circumstances was simply unprofessional. No opinion testimony based on this comment will be allowed.

This Opinion is unsupported by the facts, is based on unreliable, unscientific and (and most-likely inadmissible) evidence, and was essentially abandoned by Sottile during his deposition. Sottile's opinion regarding the length of warning time provided by the active warning system at the intersection prior to the collision is not the product of reliable methods. It is neither legally nor scientifically sufficient to meet the standards of *Daubert,* and it is therefore barred.

***10** Sottile's third opinion is that the test records for the crossing signal system "do not meet the federal requirements outlined in 49 C.F.R. § 234.273." This Regulation provides that results of inspections and tests are to be recorded in a particular way, signed by the employee, and "retained until the next record for that test is filed but in no case for less than one year from the date of the test." *Id.*

Sottile did not have access to the actual tests that were conducted on the locomotive in question because more than one year had passed and subsequent tests had been performed. This litigation was initiated more than one year after the accident. To the extent his "opinion" is critical of BNSF's failure to retain the actual test and inspection documents, his opinion is disregarded. BNSF was not required to and did not keep the actual test results. The summary of the tests and inspections from the time of the accident showed no problems or malfunctions of any part of the warning system.

Sottile also had access to the 2011 tests and inspections, which he criticized for lack of a signature and a missing decimal point on a battery test. These "deficiencies" were not reflective of malfunction of the event recorder or the warning system. Sottile himself testified in his deposition that if he had done an inspection and found the 2011 records, the deficiencies might have been criticized, but there would have been no warning issued to the railroad as there would be for serious problems.

Sottile does not dispute that this opinion has nothing whatsoever to do with the cause of the accident in question, but claims in his deposition that the deficiencies show "a culture, a pattern." (p.129–32). While there may be some situations in which deficient records would tend to show a culture or pattern of carelessness, a clerical error and a missing signature do not even come close to that level. These "deficiencies" are of no consequence in determining any issue in this action. Fed.R.Evid. 401(b). Sottile's opinion on this question is not relevant and is therefore barred.

Finally, Sottile opines that the signal event recorder data is insufficient to determine the speed, horn and active warning systems at the time of the accident, because (1) the data log is in "txt" format and can be manipulated because it was not encrypted; and (2) the data log time stamp does not reflect daylight savings time. The second question raises the same non-issue as it did above. Standard time and daylight savings time can be synchronized without the need for any assumption whatsoever, and failure to make that synchronization is simply ridiculous. No more need be said on that issue.

With respect to his criticism that the data log might have been manipulated because of its format, this is pure speculation. The data log does not contain the information Sottile expected to see, so he guessed at a couple of possible reasons. When confronted, he stated that he had no evidence of any manipulation (p.135) and no reason at all to criticize the technician who downloaded the data. (p.139–140). He admitted that he had not seen certain documents before his deposition and that the reason he did not see the expected information was that the train remained in the intersection ("on the island") until after the download of data was completed. Because the event recorder is "event driven" and no "event" occurred from the time the train stopped until the download, no more data was recorded.

***11** Once again, Sottile's opinion was based on incomplete information. Once he was shown additional documents, he back-pedaled and changed his opinion. He cannot be allowed to testify to an opinion that he has abandoned.

CONCLUSION

For the reasons stated herein, the Motions to Bar [# 56][# 58] are GRANTED in their entirety. The testimony of Mr. Bodnar and Mr. Sottile is barred.

Parallel Citations

88 Fed. R. Evid. Serv. 1321

Footnotes

1 An "event recorder" is a device that "(1) records train speed, hot box detection, throttle position, brake application, brake operations, and any other function the Secretary of Transportation considers necessary to record to assist in monitoring the safety of train operation, such as time and signal indication; and (2) is designed to resist tampering." 49 U.S.C. § 20137.

2 His lack of availability was due to his annual out-of-the country ski trip, related travel, and subsequent activity on other cases. The Order denying his motion found that this did not constitute "due diligence" that would support an extension of the deadlines in any event.

3 Actually, there is some merit to the argument that Bodnar lacked the qualifications to offer his testimony about the horn data. He did not know what type of event recorder was being downloaded or what software was used to do so, and he relied on an undisclosed "partner" to double check his work. He also applied an inapposite federal regulation. But because his opinion is based solely on one row of missing data on that download (and not on a complete reading of the entire download), that serious lack in his qualifications really does seem to go more to the weight or lack thereof of his opinion than to his qualifications. Because his opinions themselves are so far below the requisite *Daubert* standard, I do not find it necessary to reach a definitive decision on this matter.

4 Actually, what Sottile said was that the 30 seconds of warning came one hour before the accident, because he refused to make the adjustment for daylight savings time. As discussed above, that is absurd and is ignored.

EXHIBIT 12

2011 WL 2633842
Only the Westlaw citation is currently available.
United States District Court,
N.D. Illinois,
Eastern Division.

UNITED STATES of America, Plaintiff,
v.
David VANCE, Defendant.

No. 07 CR 0351. | July 5, 2011.

Opinion

MEMORANDUM OPINION AND ORDER

JOAN B. GOTTSCHALL, District Judge.

***1** The United States of America (the "government") has moved *in limine* to bar the testimony of Larry M. Dehus, the defense's proposed expert witness. For the reasons set forth below, the motion is granted.

I. BACKGROUND

David Vance is charged with committing two armed bank robberies. Vance's attorneys hired Dehus to evaluate certain discovery materials "with respect to crime scene processing procedures, proper evidence handling, sampling and testing procedures, and the degree to which all potential evidence had been evaluated." (United States' Mot. *In Limine* to Bar Defense Expert Testimony Ex. A, ECF No. 213.) These discovery materials included reports completed by the Chicago Police Department and the Illinois State Police Laboratory, photos and sketches of the crime scene, witness statements, security camera videos, and "other various materials." (*Id.*) Dehus formed the following opinions about the procedures for evidence collection and analysis followed in the investigation of the bank robberies in which Vance is alleged to have been involved:

A) The crime scenes are processed by two different agencies. This appeared to result in a lack of coordination in the collection and submission of the evidence coming from multiple sources. Based upon the lack of coordination, labeling and continuity, it is not possible to determine if the chain of custody has been compromised for many of the evidence items.

B) Latex Gloves—This examiner observed numerous problems with respect to the collection, documentation, sampling, and handling of these gloves. These current concerns are as follows:

1. The latex gloves were improperly collected in that as many as ten gloves were placed together in a single package as opposed to being properly packaged separately.

2. The lack of due care in the collection and handling of the glove evidence is evidenced by the discrepancy in the actual number of gloves being submitted. In one exhibit, The Chicago Police Department said there were 12 latex gloves and the Illinois State Police Lab reported that there were 13. In a second exhibit, a discrepancy report states that 9 latex gloves were reported as submitted, but 10 were actually received by the Illinois State Forensic Police Lab.

3. This examiner did not find documentation in either photographs or sketches to show the exact location where each of these gloves were found and collected. Photographs should have been taken of all of the gloves as found and then photographic documentation [sic] to show their collection and preservation.

4. The gloves were submitted with various types of trace evidence materials that were described but never evaluated.

5. The sampling of the gloves for DNA testing was improper. Most of the gloves were sampled using the same swab to sample the interior and exterior of gloves together. The exterior and interior of the gloves should always be sampled separately for obvious reasons.

***2** C) There is no indication that all of the clothing of Tramaine [sic] Gibson had been tested with respect to gunshot evidence or other trace evidence.

D) There is no indication that the clothing of the two security guards had been tested with respect to gunshot evidence or other trace evidence.

E) A bullet was reported to have been recovered from Dorothy Sanders on June 4, 2007, at Advocate Christ Hospital. There was no indication as to the time that it was recovered or the [sic] proper record keeping regarding a chain of evidence. Notations indicate that Det. M.L. Frasier got the bullet from Gerri Wetzel in the

Pathology Department at Advocate Christ Hospital on that date.

F) A large number of evidence items had been recovered from the 1991 Olds Delta 88 and this list includes: swabs from the steering wheel, swabs from the armrest, vacuuming samples from the interior, floor mats, contents of rear ashtray, torn material from a right rear fender. There is no indication that trace evidence from any of these items had been evaluated.

G) The proper accounting of evidence items is important to insure its [sic] integrity. An Illinois State Police Lab discrepancy notification concerning latent print lifts filed 8–28–10, more than three years after the evidence was collected, demonstrates extremely poor accountability for the evidence.

It is the opinion of this examiner that the evidence collection procedures, documentation, and processing of the evidence do not comply with standard forensic science procedures. This has resulted in evidence being lost, contaminated, and not completely evaluated.

(*Id.*) The above constitutes substantially all of the Dehus' report, which is laid out in a three-page letter dated March 24, 2011. Dehus does not explain how he reached these conclusions, other than to note that "[t]he above is a summary of my findings that were noted with respect to crime scene processing, evidence collection, evidence submission, and evidence evaluation after a review of voluminous materials in this matter." (*Id.*) According to Vance, Dehus will "testify about proper methodology and procedures for the collection, documentation and processing of evidence and will conclude that in the David Vance case, the collection procedures, documentation, and processing of evidence did not comply with standard forensic science procedures." (*Id.*)

II. LEGAL STANDARD

Federal Rule of Evidence 702 governs the admission of expert testimony. Rule 702 provides:

> If scientific, technical, or otherwise specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

***3** Fed.R.Evid. 702. Thus, expert testimony is not admissible unless it is relevant and reliable. *Daubert v. Merrell Dow Pharms ., Inc.,* 509 U.S. 579, 589, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993); *Ervin v. Johnson & Johnson, Inc.,* 492 F.3d 901, 904 (7th Cir.2007). Expert witnesses must have the "knowledge, skill, experience, training, or education" to qualify as an expert; the methodology underlying the expert's testimony must be reliable; and the expert's testimony must help the trier of fact understand the evidence or determine a fact at issue. *Myers v. Ill. Cent. R.R. Co.,* 629 F.3d 639, 644 (7th Cir.2010); *Ervin,* 492 F.3d at 904; Fed.R.Evid. 702; *see Smith v. Ford Motor Co.,* 215 F.3d 713, 721 (7th Cir.2000) ("[I]n order for an expert's testimony to qualify as 'relevant' under Rule 702 it must assist the jury in determining *any* fact at issue in the case."). In determining whether the expert's methodology is reliable, the court may consider "(1) whether the theory has been tested; (2) whether the theory has been subjected to peer review and publication; (3) the known or potential rate of error; and (4) whether it has been generally accepted within the relevant scientific community." *Happel v. Walmart Stores, Inc.,* 602 F.3d 820, 824 (7th Cir.2010) (*citing Daubert,* 509 U.S. at 593–94.).

Also relevant to the government's motion is Federal Rule of Evidence 403 and a portion of Federal Rule of Criminal Procedure 16. Rule 403 provides:

> Although relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence.

Fed.R.Evid. 403. In addition, Rule 16[1] "says that if (at defense request) the prosecution discloses details of expected expert testimony, then the defense must do so too, revealing 'the witness's opinions, the bases and reasons for those opinions, and the witness's qualification [s]'." *United States*

v. Rettenberger, 344 F.3d 702, 706 (7th Cir.2003). "The rule requires 'a summary of the expected testimony, not a list of topics.' " *Id.* (quoting *United States v. Duvall,* 272 F.3d 825, 828 (7th Cir.2001)). "The level of detail of this summary depends on the complexity of the expert testimony." *United States v. Caputo,* 382 F.Supp.2d 1045, 1049 (N.D.Ill.2005) (citing *United States v. Jackson,* 51 F.3d 646, 651 (7th Cir.1995)). Accordingly, expert witness testimony involving "technical or scientific evidence[ ] may require greater disclosure [than testimony based on experience], including written and oral reports, tests, investigations, and any other information that may be recognized as a legitimate basis for an opinion under [Federal Rule of Evidence 703]." *Jackson,* 51 F.3d at 621. In addition, the Advisory Committee notes explain that this rule is "intended to minimize surprise that often results from unexpected expert testimony, reduce the need for continuances, and to provide the opponent with a fair opportunity to test the merit of the expert's testimony through focused cross-examination." Fed.R.Crim.P. 16 advisory committee's notes.

III. ANALYSIS

A. Dehus' Disclosure Does Not Establish that He Has the Qualifications to Testify as an Expert.

***4** Vance's disclosure—which consists of the report described above, a letter from Vance's attorney summarizing Dehus' findings, and Dehus' curriculum vitae—does not adequately describe Dehus' qualifications as required by Federal Rule of Criminal Procedure 16(b)(1)(C). As a result, Vance has not established that Dehus has the "knowledge, skill, experience, training, or education," Fed.R.Evid. 702, to qualify as an expert witness on "crime scene processing procedures, proper evidence handling, sampling and testing procedures, and the degree to which all potential evidence had been evaluated." (United States' Mot. *In Limine* to Bar Defense Expert Testimony Ex. A, ECF No. 213.) According to Dehus' curriculum vitae, Dehus earned his bachelor's degree in biology (with minors in chemistry and psychology) from Otterbein College in 1965, earned his master's degree in "biology-biochemistry" at Wright State University in 1974; has been trained in forensic microscopy, forensic serology, accident reconstruction, microscopy of hairs, forensic geology, DNA analysis, the cause and origin of fires and explosions, and "BAC DataMaster" (which appears to have something to do with field sobriety testing); worked as a criminalist in a crime lab for three years, then as a technical supervisor in a crime lab for seven years; taught courses on criminalistics, forensic sciences, and criminal justice; and is a member of two professional organizations for forensic scientists, and has served as a consultant for 28 years (24 years of which he spent testing criminal evidence for defense attorneys and prosecutors). (United States' Mot. *In Limine* to Bar Defense Expert Testimony Ex. B, ECF No. 213.) In addition, Dehus published an article entitled "The Collection and Analysis of Physical Evidence from Sexual Assault Victims" in 1980. (*Id.*)

It is clear that some of Dehus' experience is too dated to qualify him to opine on current procedures for evidence collection and analysis. More often, Dehus does not indicate when he obtained certain experience, leaving the court unable to determine whether and how Dehus has stayed current with the procedures for evidence collection and analysis. In addition, Dehus does not define "forensic microscopy", "forensic serology", "microscopy of hairs", "forensic geology", or what he means by "DNA analysis," let alone how training in these areas qualifies him to opine as he does about proper procedures for maintaining a chain of custody, collecting and analyzing ballistics evidence, or collecting DNA evidence or gunshot residue from physical evidence such as latex gloves, the interior of a car, or clothing. Although Dehus indicates that he worked in a crime lab at some point, he does not indicate the skills he obtained from that position or whether he had specific experience collecting and analyzing ballistics evidence, DNA evidence or gunshot residue, or maintaining a chain of custody.

The paucity of information conveyed about Dehus' pertinent qualifications is surprising given that Dehus claims to have testified over 1500 times as an expert witness, including in federal court. In sum, because Vance's disclosures do not adequately describe Dehus' qualifications as required by Federal Rule of Criminal Procedure 16(b)(1)(C), the court cannot discern whether Dehus has the depth or recency of experience that would qualify him to testify as an expert on proper procedures for the collection and analysis of evidence. While the government's motion to exclude Dehus' expert testimony could be granted on this basis alone, the court will address other deficiencies for the sake of completeness.

B. Dehus' Disclosure Does Not Establish that his Methodology Is Reliable.

***5** In addition, the court cannot conclude that Dehus' methodology is reliable since Dehus did not adequately describe the bases and reasons for his opinions as required by Federal Rule of Criminal Procedure 16(b)(1)(C). As an initial

matter, Dehus reveals only some of the materials he relied on in forming his opinions-reports completed by the Chicago Police Department and the Illinois State Police Laboratory, photos and sketches of the crime scene, witness statements, and security camera videos. (United States' Mot. *In Limine* to Bar Defense Expert Testimony Ex. A, ECF No. 213.) His statement that he also relied on "other various materials" is not helpful. (*Id.*). Indeed, "[t]he soundness of the factual underpinnings of the expert's analysis and the correctness of the expert's conclusions based on that analysis are factual matters to be determined by the trier of fact ...." *Smith,* 215 F.3d at 718. However, the jury cannot determine whether the factual underpinnings of Dehus' opinions are sound if Dehus does not disclose all of the materials on which he relied in forming his opinions. Moreover, the government cannot properly prepare for cross-examination or procure rebuttal evidence without proper notice of the materials on which Dehus is basing his opinions.

In addition, Dehus does not reveal what methods he used to reach his conclusions or what manuals or other reference materials, if any, he relied upon, other than his noting that he reviewed voluminous materials. (*Id.*) The court is left to wonder whether he simply read certain materials and weighed the evidence, which would be improper. *See Noller v. London & Lancashire Indem. Co. of Am.,* 103 F.2d 622, 623 (7th Cir.1939) ("The jurors are the sole and exclusive judges of the facts, of the credibility of the witnesses, and of the weight of the evidence.") Vance argues that Dehus' opinions are based on his experience, but nowhere was this indicated in Dehus' report, nor does Dehus hint at the nature of the experience on which he relied. While it is true that "[a]n expert's testimony is not unreliable simply because it is founded on his experience rather than data," *Metavante Corp. v. Emigrant Savings Bank,* 619 F.3d 748, 761 & n. 8 (7th Cir.2010) ("Criminal cases, for instance, are replete with examples of experts, such as police officers or informants, qualified by experience."), as explained above, Dehus has not established that he has the requisite experience to qualify as an expert on the proper procedures for evidence collection and analysis. Had Dehus shown that he possesses the relevant qualifications, perhaps he could have assisted the jury by testifying about what is currently accepted as proper procedure for evidence collection and analysis. However, since Dehus has not shown that he has the relevant expertise, the court cannot conclude that Dehus' testimony would do anything other than invade the province of the jury. Indeed, allowing Dehus to testify based on nothing more than his review of certain discovery materials could give the jury the impression that he did something more than simply review the materials, which the jury can do itself. *See United States v. Hall,* 93 F.3d 1337, 1343 (7th Cir.1996) ("Unless the expertise adds something, the expert at best is offering a gratuitous opinion, and at worst is exerting undue influence on the jury that would be subject to control under Rule 403.")

***6** Accordingly, even if Dehus' summary had adequately described his qualifications and opinions, his report and testimony would have been excluded for failure to adequately describe the bases and reasons for his opinions.

C. Dehus Has Not Established that his Proposed Testimony Would Help the Jury Understand the Evidence or Determine a Fact in Issue.

"In determining whether expert testimony will be helpful to the jury in a particular case, the court is required to evaluate 'the state of knowledge presently existing about the subject of the proposed testimony' in light of its 'appraisal of the facts of the case.' " *United States v. Brown,* 7 F.3d 648, 651–52 (7th Cir.1993) (quoting *United States v. DeSoto,* 885 F.2d 354, 359 n. 3 (7th Cir.1989)). "Expert testimony is not admissible under Rule 702 if it will not assist the jury in understanding the evidence or determining a fact in issue or it is purely speculative." *United States v. Davis,* 772 F.2d 1339, 1333–43 (7th Cir.1985) (citing *United States v. West,* 670 F.2d 675, 682–83 (7th Cir.1982)).

It is unclear whether several of Dehus' opinions would "assist the trier of fact to understand the evidence or determine a fact in issue" as required by Federal Rule of Evidence 702. For example, Dehus opines that "[t]here is no indication that all of the clothing of Tramiane [sic] Gibson had been tested with respect to gunshot evidence or trace evidence." (United States' Mot. *In Limine* to Bar Defense Expert Testimony Ex. A, ECF No. 213.) Yet, since Dehus does not indicate the specific materials on which he based this opinion, the court cannot determine whether Dehus merely read a note on a sheet of paper that said "not tested" or whether Dehus decoded an abbreviation that the average juror would find unintelligible. In addition, Dehus is of the opinion that the investigators did not exercise "due care" since one document said that there were 12 latex gloves collected while another said there were 13. Noting discrepancies in documentation is within the understanding of the average juror. The jury, not an expert, should be charged with judging whether such discrepancies meant that the investigators did not use "due care."

Accordingly, even had Vance's disclosure adequately described Dehus' opinions, qualifications, and the bases and reasons for those opinions as required by Federal Rule of Criminal Procedure 16(b)(1) (C), several of Dehus' opinions would have been excluded because they do not assist the jury.

III. CONCLUSION

Dehus' report is totally conclusory and completely inadequate to inform the court of: (1) the knowledge, skill, experience, training or education on which he relied in forming his opinions; (2) the methodology, if any, he utilized in reaching his opinions and (3) in some cases, such as the lack of certain testing, whether he will be telling the jury anything that it cannot easily discern itself. He has not indicated whether he is relying on any scientific theory, whether any such theory has been tested and whether it has been subjected to peer review and publication, among other things. Moreover, he has not indicated whether, if the evidence collection procedures were improper (such as placing 10 gloves in one package rather than in 10 packages or failing to evaluate certain trace evidence), there is any consequence in terms of the reliability of the evidence being offered by the government. Dehus' report contains no opinions about any such consequence of the government's allegedly improper evidence-gathering techniques, raising issues about his testimony's relevance. If the government's evidence collecting methods impair the reliability of the government's evidence, not only does Dehus' report fail to so indicate, but no basis for any such opinion is given, rendering his testimony inadmissible under Rule 702, Rule 16, and *Daubert*. Without this information, the court cannot do its job as gatekeeper and the government cannot prepare to meet Dehus' testimony. Given the foregoing, the government's motion to bar Dehus' testimony is granted.

Footnotes

1 Federal Rule of Criminal Procedure 16(b)(1)(C) provides:

> The defendant must, at the government's request, give to the government a written summary of any testimony that the defendant intends to use under Rules 702, 703, or 705 of the Federal Rules of Evidence as evidence at trial, if—
> (i) the defendant requests disclosure under subdivision (a)(1) (G) and the government complies; or
> (ii) the defendant has given notice under Rule 12.2(b) of an intent to present expert testimony on the defendant's mental condition.
> This summary must describe the witness's opinions, the bases and reasons for those opinions, and the witness's qualifications.

End of Document

EXHIBIT 13

2008 WL 656045
Only the Westlaw citation is currently available.
United States District Court,
N.D. Illinois,
Eastern Division.

MURATA MANUFACTURING CO., LTD., Plaintiff,
v.
BEL FUSE, INC., Bel Fuse, Ltd., Bel Stewart, Ltd., and Bel Connector, Inc. d/b/a Stewart Connector and Bel Stewart, Defendants.

No. 03 C 2934. | March 5, 2008.

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Opinion

MEMORANDUM OPINION AND ORDER

JOAN B. GOTTSCHALL, District Judge.

***1** Plaintiff Murata Manufacturing Co., Ltd. ("Murata") has filed a motion to strike and bar portions of defendants Bel Fuse, Inc. et al .'s (collectively "Bel") brief in opposition to Murata's motion for summary judgment of literal infringement of Murata's U.S. Patent No. 5,069,641 ("the #641 patent"). Specifically, Murata seeks to strike five allegedly new theories of noninfringement that it claims Bel raises for the first time in its brief in opposition, and to further bar Bel from raising those theories at trial. Murata additionally seeks to strike the declarations of Bel's expert witnesses R. Lee Hill ("Hill") and Peter G. Bittner III ("Bittner") which were submitted as exhibits accompanying Bel's brief in opposition. For the reasons set forth below, Murata's motion is granted in part and denied in part.

I. ANALYSIS

1. First Alleged Theory of Noninfringement:
In response to Murata's motion to strike, Bel has withdrawn any argument, in its brief or at trial, based upon the first allegedly novel theory of noninfringement; the reverse doctrine of equivalents. *See, e.g., Tate Access Floors, Inc. v. Interface Architectural Res., Inc.,* 279 F.3d 1357, 1368 (Fed.Cir.2002). The court therefore strikes that theory of noninfringement from both Bel's opposition to Murata's motion for summary judgment of literal infringement and at trial.

2. Second Alleged Theory of Noninfringement:
Murata argues that Bel also presents a new theory of noninfringement with respect to the Family 1 modular jacks, *viz.,* that the contactors are not "electrically connected" to the noise suppressing elements because the contactors engage pins in a toroid base which, in turn, fit into holes in the printed circuit board, engaging the traces on the board. Murata's Mot. to Strike § II ¶ 2. Bel retorts that its statement in opposition to Murata's motion to dismiss claims that "the Family 1 connectors still do not infringe because they lack a contactor being electrically connected with the electronic element 'by a wire on the printed board' as required by the claims." Bel's Opp. at 13. Bel cites the expert report submitted by Hill during discovery, which claims that "the capacitor is not 'electrically connected' to any contactor by a 'wire on the printed board.' Rather, the contactors are connected to the resistors or to other components. Bel's Brief in Opp. Ex. 2 ¶ 55. Hill's report thus clearly indicates that the connection between the contactor and the capacitor is not a direct contact mediated only via a "wire on the board," but that "other components" intervene. *Id.* Even a cursory glance at the illustrations of the Family 1 components at issue provided in both parties' briefs renders this argument apparent.

Murata replies, cryptically, that Bel's explanation of its argument is not more precise, but rather more abstract. Murata's Reply Brief 7-8. Furthermore, it claims that because the phrase "other components" appears, Bel can then continue to plug any component it wants into its defense theory. *Id.* at 8. Murata claims that Bel's factual submissions and arguments therefore violate Federal Rule of Civil Procedure 26(a)(2)(B), which requires disclosure in expert reports of "a complete statement of all opinions to be expressed and the basis and reasons therefore." *Id.* at 8. Murata complains

that the Hill report says nothing about "intermediate pins" or "toroid bases" despite its alleged requirement to do so. *Id.*

***2** Murata's argument, however, is a canard; Hill's report states that there is not a direct connection from contactor to "wire on the board" to capacitor, but rather that another component intervenes and thus the accused device lies outside the scope of the claims. Bel is not required to list every possible permutation of potential components; rather, its argument is that the connection is not direct, but indirect. That much is clear from Hill's report, and a straightforward reading of the report should have put Murata on notice of Bel's argument in this respect. Moreover, Murata's invocation of *Salgado v. General Motors Corp.* avails it little. 150 F.3d 735 (7th Cir.1998). *Salgado* describes the requirement that an expert's report must be sufficiently "detailed and complete" so that opposing counsel will not be ambushed at trial. 150 F.3d at 741 n. 6. Hill plainly asserts that there is no connection between the contact and the capacitor via a "wire on the board", but rather a connection between the contacts and resistors and other components. Bel's Brief in Opp. Ex. 2 ¶ 55. The court finds that Hill's argument is sufficiently detailed and complete, and supported by facts, so as to adequately satisfy the requirements of Rule 26(a)(2)(B).

Nor is Murata's argument that Hill's prior report is contradicted by the new report convincing. The court's understanding of electronics is not so unsophisticated as to fail to comprehend the difference between "connected" (implying a physical contact connection) and "electrically connected" (which implies existence of a conductive pathway through which current may flow from one component to another [1]). The court therefore finds that Bel adequately disclosed the facts underlying this particular argument and denies Murata's motion to strike this particular theory of noninfringement.

3. Third Alleged Theory of Noninfringement:

Next, Murata argues, also with respect to Family 1, that the contactors are not connected via a "wire on a printed board" to noise suppressing elements because "the terminals are connected to wires wound around toroids and only part of this path is a trace." Murata's Mot. to Strike § II ¶ 3.

Bel counters with the argument that "the Family 1 connectors do not infringe because they lack a terminal that is 'electrically connected' to the electronic element 'by a wire on the printed board' as required by the claims." Bel Opp. at 16. Bel's expert report by Hill states explicitly that none of the contactors "are 'electrically connected' to any of the terminals ... because of the presence of the isolation transformers." Brief in Opp. Ex. 2 ¶ 60. Bel contends that the presence of the isolation transformers precludes any electrical connection by a wire on the board due to the electrical isolation created by the wire wrapped toroids in the base. Bel Opp. at 17.

Murata argues in reply that Bel's expert report comprises a novel theory of noninfringement that is related to an entirely different claim limitation ("electrically connected") in addition to prior theories that were related to the limitation "by a wire on the board." Murata Reply Brief 10.

***3** The court finds that the plain language of Hill's report corresponds sufficiently to Bel's argument in its opposition to Murata's motion for summary judgment to satisfy the requirements of Federal Rule of Civil Procedure 26(a)(2)(B). Murata's motion with respect to this argument is consequently denied.

4. Fourth Alleged Theory of Noninfringement:

Next, Murata argues that, with respect to Family 3, Bel's contention that the #641 patent "indicates" that the substrate in a chip is not a printed board is a new and impermissible noninfringement theory. Murata's Mot. to Strike § II ¶ 4.

Bel counters by arguing that the distinction between a printed board and a chip has been explicitly presented in Hill's report. Brief in Opp. Ex. 2 ¶ 114 ("I have never to my recollection ever read, heard, or even conceived of the notion that the substrate of a chip resistor could be considered by anyone in the electronics industry as constituting a printed board"). Bel argues that Murata's new argument, raised in its motion for summary judgment, is that the chip resistor includes a printed board, and that their argument in opposition constitutes a proper rebuttal. Brief in Opp. 10 (citing *Aircraft Gear Corp. v. Marsh,* No. 02 C 50338, 2004 WL 1899982, at *5 (N.D.Ill. Aug.12, 2004).

Murata retorts that Bel's argument in opposition to Murata's motion for summary judgment is the very first time that Bel has argued that there is a "clear line of demarcation" between printed boards and substrates in chip resistors, and argues that Bel has never before made that argument. Murata Reply Brief 10. Murata argues that Bel has moved for summary judgment based on this differentiation between printed boards and chip resistors.

Hill's statement, however, explicitly states that, as an expert, he believes that there is a cognizable dichotomy between chip resistors and printed boards. Murata argues that "determination of what a patent teaches is fact finding, not interpretation." *Id.* at 11 (citing *Graham v. John Deere Co. of Kansas City,* 383 U.S. 1, 17, 86 S.Ct. 684, 15 L.Ed.2d 545 (1966)). Nevertheless, it is precisely such a factual inquiry that is the function of this court, and Hill's opinion, explicitly stated in his report, stakes out a dispute over that factual inquiry that is fairly included in Bel's opposition. Murata's motion to strike this portion of Bel's opposition is denied.

5. Fifth Alleged Theory of Noninfringement:

Finally, Murata has submitted a final argument in its supplement to its motion to strike. Murata contends that Bel's claim that the alumina substrate layer of the chip resistor does not constitute a printed board (and therefore does not infringe the #641 patent) is a new theory of noninfringement. Murata argues that this argument was not supported by expert testimony, and that it was only raised in Bel's response in opposition to Murata's motion for summary judgment. Supp. to Murata's Mot. to Strike 2. Murata claims that Bel's theory relies solely on dictionary definitions that were supplied by Bel only at the last minute.

***4** In response, Bel cites explicit testimony from Hill's report that the alumina substrate layer does not contain a plurality of the electrically interconnected components that this court construed to be elements of a printed board in its *Markman* hearing.[2] Brief in Opp. Ex. 2 ¶ 114; *Murata Mfg. Co., Ltd. v. Bel Fuse Inc.,* 445 F.Supp.2d 938, 947 (N.D.Ill.2006). As such, it is evident that Murata could have deposed Bel's expert on that explicit statement during discovery. Moreover, Bel advanced this theory of infringement in its response brief (and not a reply brief, as alleged by Murata), to Murata's motion for summary judgment of literal infringement. Murata has consequently had the opportunity to rebut Bel's argument in its reply brief and will again have that opportunity at trial. Murata's motion to strike the argument and bar it from further proceedings is consequently denied.

6. The Declarations of Bittner and Hill

Murata also opposes the submission of two affidavits, by Hill and Bittner respectively, submitted by Bel with its brief in opposition to Murata's motion for summary judgment. According to Murata, Bel is attempting to use these declarations to support the allegedly new theories of noninfringement submitted in its opposition to Murata's motion for summary judgment. Murata's Mot. to Strike § III. Murata complains that because the affidavits were submitted after the close of discovery, it had no opportunity to depose Hill or Bittner on the facts alleged in their affidavits concerning their contentions of noninfringement. Murata cites no legal support for their motion to strike the affidavits and their argument has little merit.

Rule 56(e) of the Federal Rules of Civil Procedure explicitly permits the submission of affidavits in conjunction with, or in opposition to, a motion for summary judgment. Fed.R.Civ.P. 56(e). Moreover, the Rule requires that an "opposing affidavit must be made on personal knowledge, set out facts that would be admissible in evidence, and show that the affiant is competent to testify on the matters stated." *Id.* These requirements are mandatory. *Toro Co. v. Krouse, Kern & Co., Inc.,* 827 F.2d 155, 162 n. 3 (7th Cir.1987).

An affidavit submitted in conjunction with a brief in opposition to a motion for summary judgment must be limited to facts, and the facts must be alleged on personal knowledge. Fed.R.Civ.P. 56(e); *In re Morris Paint and Varnish Co.,* 773 F.2d 130, 135-36 (7th Cir.1985) (citing *Ashwell & Co. v. Transamerica Ins. Co.,* 407 F.2d 762, 766 (7th Cir.1969)). Thus, ultimate or conclusory facts and conclusions of law, as well as statements made on belief, may not be employed in a summary judgment motion. *See Corder v. Lucent Technologies Inc.,* 162 F.3d 924, 927 (7th Cir.1998); *Resolution Trust Corp. v. Juergens,* 965 F.2d 149, 152-53 (7th Cir.1992). However, expert witnesses, who are not strictly "fact witnesses," may opine in their affidavits, provided they do more than offer mere naked conclusions; they must also provide a process of reasoning underlying those conclusions, beginning with a firm foundation. *Mid-State Fertilizer Co. v. Exchange National Bank of Chicago,* 877 F.2d 1333, 1339 (7th Cir.1989) ("An expert who supplies nothing but a bottom line supplies nothing of value to the judicial process."); *See also Zarecki v. National R.R. Passenger Corp.,* 914 F.Supp. 1566, 1575 (N.D.Ill.1996).

***5** Finally, the facts contained within the affidavits must be admissible as evidence at trial. *Reeves v. Commonwealth Edison Co .,* No. 06 C 5540 2008 WL 239030 (N.D.Ill. Jan. 28, 2008). The admissibility of expert testimony is governed by Federal Rule of Civil Procedure 702 and the U.S. Supreme Court's decision in *Daubert v. Merrell Dow Pharmaceuticals,*

Inc., 509 U.S. 579, 591, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993).

Under *Daubert,* this Court must conduct a two-step analysis to determine whether an expert's opinion is admissible. First the court must determine whether the expert's testimony pertains to scientific, technical, or other specialized knowledge. *Kumho Tire Co., Ltd. v. Carmichael,* 526 U.S. 137, 141, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999). The district court must consider whether the testimony has a reliable basis in the in the knowledge and experience of the discipline; it must rule out subjective belief or unsupported speculation. *Kumho,* 526 U.S. at 149 (citing *Daubert,* 509 U.S. at 590). Second, the district court must "determine whether the evidence or testimony assists the trier of fact in understanding the evidence or in determining a fact in issue. That is, the suggested expert testimony must 'fit' the issue to which the expert is testifying." *Porter v. Whitehall Lab.,* 9 F.3d 607, 616 7 th Cir.1993). Essentially, purpose of the rule in *Daubert* is "to make sure that when [scientists/ engineers/technical experts] testify in court they adhere to the same standards of intellectual rigor that are demanded in their professional work." *Rosen v. Ciba-Geigy Corp.,* 78 F.3d 316, 318 (7th Cir.1996).

The court finds that there is sufficient factual foundation, presented on firm and accepted technical footing, to support the opinions and conclusions set forth in Hill's and Bittner's affidavits. Both Hill and Bittner appear from their resumes and affidavits to be competent to provide expert testimony, and the scientific (engineering, to be precise) bases forming the underpinnings of their opinions and conclusions appear to meet the standards required by *Daubert* and *Kumho* [3] for admissibility as expert testimony. Moreover, the subject matter that both affiants opine upon is material to the case and likely to assist the court in understanding the evidence or in determining the factual issues before it at trial. Thus, both prongs of the *Daubert* analysis are satisfied and Hill's and Bittner's testimony are admissible as evidence. Murata's motion to strike and bar the affidavits of Hill and Bittner is consequently denied.

II. CONCLUSION

For the reasons set forth above, Murata's motion to strike and bar Bel's noninfringement theories and expert affidavits is granted with respect to Bel's theory based upon the reverse doctrine of equivalents, and denied with respect to the rest of its motion.

Footnotes

1 Although to remain within the scope of the claims, the connection through which the current flows must include a "wire on the board."

2 In its *Markman* hearing, the court construed "printed board" as "a generally flat piece of material typically fabricated from insulating material that provides support and structural integrity for a plurality of electrically interconnected components comprising a circuit, with some or all of the conducting interconnection pattern formed on the board." *Murata,* 445 F.Supp.2d at 947.

3 In *Daubert,* the Court identified several factors that should be considered when determining whether testimony has been subjected to the scientific method: (1) whether the theory can be and has been tested; (2) whether the theory has been subject to peer review and publication; (3) the known or potential rate of error of the technique; and (4) whether the theory has been generally accepted by the relevant scientific community. *Daubert,* 509 U.S. at 593-94. The Court's conclusion must be based solely on the principles and methodology underlying the expert's conclusion, not the conclusion itself. *Id.* at 593-93. Of these four factors, the first-whether the proffered theory has been tested-has been deemed the most important. *See, e.g., Zarecki,* 914 F.Supp. at 1574; *Schmaltz v. Norfolk & W. Ry. Co.,* 878 F.Supp. 1119, 1121 (N.D.Ill.1995). The methodology and analysis performed by both Hill and Bittner reflect general and widely accepted principles of electrical engineering sufficient to ensure admissibility under these *Daubert* criteria. *See Kumho,* 526 U.S. at 141.

EXHIBIT 14

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

MARILYN F. QUIRIN, as Executor of the Estate of RONALD J. QUIRIN, Deceased, Plaintiff, v. LORILLARD TOBACCO COMPANY., *et al.*, Defendants.	Civil Action No. 13-cv-02633 Judge Joan B. Gottschall

AFFIDAVIT OF SURESH H. MOOLGAVKAR, M.D., PH.D.

Dr. Suresh H. Moolgavkar, being duly sworn, deposes and states as follows:

FURTHER AFFIANT SAYETH NAUGHT

Suresh H. Moolgavkar, M.D., Ph.D.

Subscribed and sworn before me on this 30th day of July 2013,

Notary Public

MICHELLE RANI AWASTHI
NOTARY PUBLIC
STATE OF WASHINGTON
COMMISSION EXPIRES
APRIL 21, 2014

My commission expires April 21, 2014.

INTRODUCTION

In this declaration, I discuss briefly the basic biological principles underlying carcinogenesis and describe the appropriate scientific framework for establishing cancer causation following exposure to environmental agents, such as asbestos.

1. Most chronic diseases, including cancer, have multi-factorial etiologies, i.e., may occur after exposure to one or more of a number of agents and can even occur spontaneously without exposure to any environmental agent. This fact makes the attribution of cause much more difficult than for infectious disease. For example, tuberculosis is, by definition, a disease caused by infection with the tubercle bacillus (mycobacterium tuberculosis). Once the diagnosis of tuberculosis is established, the cause of the disease is known, although the exact strain of the bacterium in any specific case may need further laboratory tests.

2. Furthermore, most chronic diseases, including cancer, develop many years after exposure (the latency period) making the attribution of cause even more difficult. For these reasons, it is important that the proper framework be developed when investigating the cause of any chronic disease, including cancer.

3. Dr. Brodkin opines that because mesothelioma has been called a sentinel disease for asbestos exposure and because it is a "dose-responsive" disease, every exposure to any type of asbestos fiber, no matter how small, is a substantial contributing factor in the causation of mesothelioma. I believe, for the reasons set forth below, that this opinion is not scientifically defensible. The scientific literature clearly demonstrates that not every type of asbestos fiber is equally potent as a mesotheliogen. In addition, there is credible evidence derived from diverse sources suggesting that mesothelioma, like all other cancers, can occur spontaneously as a result of naturally occurring biological processes without exposure to asbestos or any other environmental agent. Furthermore, numerous well-conducted epidemiologic studies have failed to demonstrate an increased risk of mesothelioma among vehicle mechanics known to be exposed to low concentrations of chrysotile asbestos, suggesting there is a level of exposure to chrysotile asbestos below which there is no detectable increase in mesothelioma risk. Moreover, there is no epidemiologic evidence that work as a vehicle mechanic increases the risk of mesothelioma imposed by other exposures to asbestos, suggesting that exposure to low levels of chrysotile asbestos does not increase the risk of mesothelioma imposed by other exposures.

4. Essentially, Dr. Brodkin argues that because exposure to high levels of commercial chrysotile contaminated with amphiboles increases the risk of developing mesothelioma, exposure to low levels of Calidria chrysotile, which is not known to be contaminated with amphiboles, also increases the risk of mesothelioma. This argument is fatally flawed as I show in the rest of this affidavit. To support his opinions, Dr. Brodkin cites highly selective literature, fails to acknowledge the limitations of the studies he cites, and fails to cite and review critically the studies that do not support his position. I will give examples of this bias in the body of this affidavit.

BACKGROUND AND QUALIFICATIONS

5. I am a physician with a Ph.D. in Mathematics and post-doctoral training in Pharmacology, Biophysics, Epidemiology, and Biostatistics. In April 2007, I became a Corporate Vice President and the Director of the Center for Epidemiology, Biostatistics and Computational Biology at Exponent, Inc., an international scientific consulting company. I retired from my position as a Full Member of the Fred Hutchinson Cancer Research Center in August 2008. I continue to be an Affiliate Investigator at the Center and Professor of Epidemiology and Adjunct Professor of Applied Mathematics at the University of Washington in Seattle. I was also Adjunct Professor of Biostatistics at the University of Washington between 1984 and 2008. I am a cancer epidemiologist and research scientist. My main research interest is cancer epidemiology. I was instrumental in developing a biologically-based mathematical model, the two-stage clonal expansion (TSCE) model, often called the Moolgavkar-Venzon-Knudson (MVK) model, for the quantitative estimation and prediction of cancer risk. This model is recognized and used by cancer researchers worldwide.

6. I have served on the faculties of the Johns Hopkins University, Indiana University, the Fox Chase Cancer Center and the University of Pennsylvania. I have been a visiting scientist at the Radiation Effects Research Foundation in Hiroshima, the International Agency for Research on Cancer (IARC) in Lyon, and the German Cancer Research Center in Heidelberg.

7. I have served on numerous review panels and as a consultant to the National Cancer Institute (NCI); the Environmental Protection Agency (EPA); the California Air Resources Board; Health and Welfare, Canada; IARC; the CIIT Centers for Health Research; and the Health Effects Institute. I am the author or co-author of more than 160 papers in the areas of Epidemiology, Biostatistics, and Quantitative Risk Assessment, and have edited three books in these areas. Among these is a monograph, "Quantitative Estimation and Prediction of Human Cancer Risk," published by IARC, the agency that conducts cancer research under the auspices of World Health Organization. I have served on the editorial boards of *Genetic Epidemiology* and *Inhalation Toxicology*. In 2012, I stepped down from my position as one of the editors of *Risk Analysis – An International Journal*, but continue to serve on the editorial board. I am an elected member of the American Epidemiological Society. I was given the Founders' Award by the CIIT Centers for Health Research in 1990, the Distinguished Achievement Award by the Society for Risk Analysis (SRA) in 2001 and the Outstanding Service Award by SRA in 2012. I am one of a few members of SRA to have received both the Distinguished Achievement and Outstanding Service Awards. I am a Fellow of SRA, the pre-eminent international scientific society for risk assessment.

8. Among my publications are several papers on carcinogenesis following exposure to fibers. I was an Invited Expert at a workshop, "Mechanisms of Fiber Carcinogenesis," held at IARC in Lyon, France, in early November, 2005. I was the lead panelist for a symposium on fiber carcinogenesis held in Brussels in 2005.

9. My employer, Exponent, Inc., charges $575 per hour for my consulting services. My curriculum vitae is attached as Appendix 1 of this affidavit.

PURPOSE OF AFFIDAVIT

10. In this affidavit, I describe the appropriate framework for examining issues of causality associated with exposure to putative carcinogens, and apply it to discuss issues of causality in mesothelioma.

11. Any framework for a discussion of causality in mesothelioma must address the following facts.

    a) Mesothelioma, like other cancers, can occur spontaneously without exposure to any environmental agents.

    b) In addition to exposure to amphibole asbestos, the risk of mesothelioma can be increased by exposure to other fibers, such as erionite, and to ionizing radiation.

    c) Independently of exposure to environmental agents such as amphibole and erionite fibers and ionizing radiation, age is a strong risk factor for the development of mesothelioma with the risk of developing mesothelioma increasing strongly with age as is true for other adult-onset cancers.

    d) There is still controversy in the literature regarding whether chrysotile asbestos uncontaminated by amphiboles can increase the risk of pleural mesothelioma.

    e) There is general consensus that amphibole asbestos is far more potent as a pleural mesotheliogen than chrysotile, if pure chrysotile increases the risk of mesothelioma at all.

    f) Numerous epidemiologic studies of the association between work as a vehicle mechanic and mesothelioma have found no increased risk, suggesting strongly that there is a level of exposure to chrysotile asbestos below which no increase in the risk of mesothelioma can be detected in well-designed epidemiologic studies.

    g) There is no epidemiologic evidence that drywall finishers not exposed to amphiboles are at increased risk of mesothelioma.

12. In any specific case, evaluation of whether a particular asbestos exposure is a substantial contributing factor requires that the background risk of mesothelioma be considered, along with all other exposures that could have increased the risk of mesothelioma in that individual. The importance of any specific exposure clearly depends on what other exposures were received. The appropriate concept from Epidemiology that needs to be applied here is that of attributable fraction (AF), which I discuss this in greater detail below.

13. I begin with a brief discussion of the biological principles underlying the process of

carcinogenesis and the role of environmental agents, such as asbestos and ionizing radiation, in modifying the rate at which this process occurs. I then discuss general principles of epidemiology and set up the framework for assessing causality. Finally, I apply this framework to mesothelioma and show that the proposition that every exposure to asbestos, no matter how small, is a substantial contributing factor to mesothelioma is not scientifically defensible.

14. I have reached my conclusions based on my knowledge as a physician, epidemiologist, and biostatistician. The basic biological principles underlying carcinogenesis have been elucidated and described in the peer-reviewed literature. A large number of epidemiologic studies published in the peer-reviewed literature have investigated the association between exposure to asbestos and the subsequent development of mesothelioma. More recent studies have evaluated the various types of asbestos fibers with respect to their relative potencies for causing mesothelioma. Other studies have specifically investigated a possible association between exposure to low levels of chrysotile exposure while working as a vehicle mechanic and mesothelioma. My opinions in this affidavit depend heavily on this entire body of literature. I hold these opinions to a reasonable degree of scientific and medical certainty. I reserve the right to revise my opinions as more information becomes available.

FUNDAMENTAL PRINCIPLES OF CARCINOGENESIS

15. It is generally recognized that cancer is the end result of an accumulation of critical mutations[1] in a cell. Mutations in a cell can and do occur spontaneously (*i.e.*, during the normal process of cell division, without any exposure to environmental agents). There are approximately 50 trillion cells in the average human body (Tomlinson et al., 2002). When a cell divides into two daughter cells, the DNA, which carries the genetic blueprint for the cell, is duplicated faithfully, with one copy being inherited by each of the daughter cells. The DNA is a very large molecule, however, consisting of some 5 billion units called base pairs.

16. Despite the existence of sophisticated cellular machinery that oversees the fidelity of DNA replication, mistakes in replication occur regularly. Once these mutations are fixed in the DNA, they are faithfully passed on to future generations of cells and accumulate over the life of an individual. Tomlinson et al. (2002) estimate that, by the age of 15 years, there are thousands of mutations in an individual human body, and this number increases with age. When a cell accumulates multiple mutations at critical gene loci, it can escape from the normal growth restraints and grow uncontrollably, to give rise to cancer (Moolgavkar and Knudson, 1981; Moolgavkar et al., 1999; Knudson, 2001). Along the pathway to cancer, a cell that has acquired some, but not all, mutations required to make it malignant may partially escape growth control and begin to replicate

[1] A mutation is an alteration of the genetic material in a cell that is passed down to daughter cells when the cell divides. Most mutations are neutral, in that they do not affect the normal functioning of the cell. However, the accumulation of mutations in critical genes can release the cell from normal growth constraints, leading to uncontrolled growth, which is the hallmark of cancer.

somewhat faster than its neighbors. Since this cell carries some of the critical mutations on the pathway to cancer, and since these mutations are passed on to its daughters when it divides, the preferential division of this cell leads to a population of cells that carry some of the mutations on the pathway to cancer. This process of clonal[2] expansion of partially altered cells on the pathway to cancer greatly increases the probability of cancer in the tissue by increasing the population of cells that carry critical mutations.

17. While the specific gene loci involved in carcinogenesis are not known for most cancers, recent work in molecular genetics has shed some light on a locus involved in some cases of mesothelioma (Testa et al., 2011). A germline[3] mutation at this locus confers a high risk of developing mesothelioma in the individual who inherits it. The hereditary transmission of this mutation explains the high incidence of mesothelioma in some families; however, it is not known how common this mutation is, so it is not currently possible to estimate quantitatively the contribution made by this germline mutation to the burden of mesothelioma in the general population. Nonetheless, the discovery of this susceptibility locus does suggest that a final common pathway for mesothelioma pathogenesis in both hereditary and sporadic (non-hereditary) cases involves mutation at the locus.

Age is a strong risk factor for the development of cancer

18. Because the process of accumulation of mutations occurs spontaneously and continuously throughout life, a certain background rate of spontaneously arising cancer must be expected in the general population. The strong impact of age on the risk of cancer has been recognized for more than 50 years (e.g., Armitage and Doll, 1954; Moolgavkar and Knudson, 1981; Meza et al., 2008; World Health Organization [WHO], 2011). For most cancer sites, including mesothelioma (Moolgavkar and Knudson, 1981; Meza et al., 2008; Moolgavkar et al., 2009), the incidence rate of spontaneously occurring cancer increases strongly with age. Thus, at age 70, the risk of pleural mesothelioma is approximately 30-fold the risk at age 35 (Moolgavkar et al., 2009).

<u>Action of environmental agents</u>

19. As described in the previous section, the current paradigm views carcinogenesis as a process of mutation accumulation, with clonal expansion of partially altered cells increasing the efficiency of the process. While, as discussed above, this process occurs spontaneously without exposure to any external agents, it can be greatly accelerated in the presence of environmental agents such as radiation, tobacco smoke, and asbestos (Moolgavkar and Knudson, 1981; Moolgavkar et al., 1999). Although the specific details are different for different environmental agents, all agents that increase the risk of cancer act, in the final analysis, in one or both of two ways: 1) by increasing the rate of mutations and/or 2) by increasing the rate of clonal expansion of partially altered cells on the pathway to cancer. Agents that increase the rate of mutations in cells are known as mutagens. Agents that increase the rate of clonal expansion of partially altered cells are

[2] A clone of cells is the population of cells consisting of a cell and all its descendants.
[3] A germline mutation is inherited from one of the parents.

known as promoters.[4] Cigarette smoke is a complex mixture that contains both mutagens and promoters. Asbestos fibers can, under certain circumstances, set up an inflammatory reaction in tissues that leads to the release of agents that act as both mutagens and promoters (Berman and Crump, 2003).

20. This brief discussion of carcinogenesis and the action of environmental agents provides a framework for understanding the concept of causal associations between exposures to environmental agents and specific cancers. An environmental agent is causally associated with a specific cancer if it increases the risk of spontaneously occurring cancer, *i.e.*, if the risk in exposed populations is larger than the risk in populations not exposed to the environmental agent. The science that investigates risks in exposed human populations is epidemiology. Thus, properly designed, conducted, and analyzed epidemiologic studies establish whether or not risk is increased in exposed populations and, therefore, provide the critical link in establishing causal associations between exposure to environmental agents and cancers at specific sites.

Case reports cannot be used to infer associations, let alone causality

21. In my experience plaintiffs' experts often cite case reports of mesothelioma among patients allegedly exposed to pure chrysotile or to low levels of asbestos to support the propositions that pure chrysotile and low levels of asbestos exposure can cause mesothelioma. Dr. Brodkin clearly relies heavily on case reports. Many of his primary citations are to case reports and some of the reviews on which he relies (e.g., Kanarek, 2011) are based heavily on case reports. Case reports can raise suspicion that a specific exposure is associated with disease; however, properly designed analytical epidemiologic studies need to be performed to establish associations between environmental exposures and disease as is discussed below. A number of analytical epidemiologic studies have been prompted by clinical reports of exposures in patients with a specific disease; however, case reports cannot by themselves establish associations, let alone causality, between the exposure and the disease because they lack a critical element of a properly designed study, namely a control group.

22. The inadequacy of case reports to establish associations between putative risk factors and disease has been recognized for many years. For example, in a well-regarded text book, Hennekens et al. (1987) state, "While case reports and case series are very useful for hypothesis formulation, they cannot be used to test for the presence of a valid statistical association. One fundamental limitation of the case report is that it is based on the experience of only one person. **The presence of any risk factor, however suggestive, may simply be coincidental.** Although case series are frequently sufficiently large to permit quantification of frequency of an exposure, the interpretability of such information

[4] The term "promotion" is used in somewhat different ways by different investigators. Here, by promotion I will mean the clonal expansion (i.e., the preferential growth) of partially altered cells on the pathway to cancer. A promoter is any agent that increases the rate of spontaneously occurring promotion. Promoters can be either endogenous or exogenous. For example, hormones are well known to be endogenous promoters of breast and prostate cancer. Cigarette smoke contains a number of agents that act as exogenous promoters in lung cancer.

is severely limited by the lack of an appropriate control group. This lack can either obscure a relationship or suggest an association where none actually exists" (emphasis added). In the same vein, in his book titled "Causal relationships in medicine: A practical system for critical appraisal," Elwood (1988) recognizes a hierarchy of evidence for the establishing of causal relationships. The best evidence is derived from randomized trials, followed by epidemiologic studies. Case series, which are a collection of case reports, are at the bottom of the list. Randomized trials clearly could not be used to investigate the toxicity of asbestos in human subjects; thus, Epidemiology is our best scientific tool to investigate the potential toxicity of asbestos in human populations.

23. In some instances, properly designed epidemiologic studies confirm the suspicion raised by case reports. The best example of this is provided by the causal association between cigarette smoking and lung cancer, which has been demonstrated in numerous epidemiologic studies. The original suspicion of such an association was raised by reports of cigarette smoking among patients with lung cancer. A more recent example is provided by vinyl chloride, which is causally associated with hepatic angiosarcoma (Bosetti et al., 2003). Other examples indicate, however, that suspicions raised by case reports are not always supported by properly conducted epidemiologic studies. Two examples are provided by the cases of Bendictin an effective anti-nausea agent during pregnancy, which was driven off the market by the suspicion that it caused birth defects, and silicone breast implants, which were reported to cause auto-immune disorders in women. In both these examples, properly conducted epidemiologic studies showed no associations between exposure to these agents and the suspected health effects (McKeigue et al., 1994; Janowsky et al., 2000).

24. Plaintiffs' experts, including often cite case reports of mesothelioma among automobile mechanics to support their contention that this occupational group is at increased risk of mesothelioma. Kanarek (2011), one of Dr. Brodkin's reliance documents, relies on case reports of mesothelioma among automobile mechanics while completely ignoring the large body of epidemiologic studies on this topic as I discuss later. The conclusion that automobile mechanics are at increased risk of mesothelioma is not justified, however, as is shown by the following example. In a 1991 letter to the editor, Woitowitz and Rödelsperger reported on cases of mesothelioma among motor mechanics in an outpatient clinic and concluded, "In summary, we have found an increased incidence of mesothelioma among car mechanics exposed to only chrysotile…" Wong (1992) pointed out that Drs. Woitowitz and Rödelsperger had not conducted a proper epidemiologic study and that no associations could be inferred from case reports. In their response to Dr. Wong's letter Drs. Woitowitz and Rödelsperger (1992) conceded that he was correct and that "…it is not possible without further information to infer an increased incidence of mesothelioma from these case figures…" Finally, Drs. Woitowitz and Rödelsperger (1994) completed a case-control study based on their preliminary observations and concluded, "From these results there is no evidence that car mechanics are exposed to an increased risk of mesothelioma if they do brake repairs…" Thus, in this example, case reports led to the hypothesis that work on brakes posed an increased risk of mesothelioma; however, a properly conducted case-control study subsequently showed no evidence of increased risk.

25. These examples illustrate that a hierarchy of evidence is used for decision-making in the biomedical and health sciences. When case reports suggest associations between a putative risk factor and disease, it may be prudent health policy to accept this observation as indicating a causal relationship in the absence of any epidemiologic evidence. As well-designed epidemiologic studies become available, however, the scientific data and results from these studies supersede the case reports. As discussed above, in some instances, the epidemiologic studies confirm the suspicions aroused by the case reports. In others, the epidemiologic studies indicate that the suspicions were unfounded. Epidemiologic studies of brake work and mesothelioma have been undertaken precisely because of the suspicion that brake workers might be at risk for mesothelioma. As discussed later in this report, not one single epidemiologic study has shown a positive association between brake work and mesothelioma. These studies span several different countries, study designs, and investigators – and all come to the same conclusion: no increased risk of mesothelioma in automobile mechanics. These same epidemiologic studies have confirmed, on the other hand, the positive association between other non-brake occupational exposures to asbestos and mesothelioma. The studies of vehicle mechanics and brake workers suggest strongly that there is a level of exposure to chrysotile asbestos, possibly contaminated with low levels of tremolite, below which no increased risk of mesothelioma can be detected in properly-designed epidemiologic studies.

Properly designed epidemiologic studies are required to establish causality between exposures and disease

26. Epidemiology is the study of diseases in populations. The science of Epidemiology investigates the distribution and determinants of diseases in populations. Epidemiology is fundamental to establishing causal relationships between exposures and disease. Epidemiology is also central to establishing quantitative exposure-response[5] relationships between exposure to an environmental agent and the risk of disease. As I discuss below, quantitative exposure-response relationships are essential in attributing risks to a specific exposure and, therefore, in determining whether or not that exposure is a "substantial contributing factor" to the risk of disease.

FUNDAMENTAL PRINCIPLES OF EPIDEMIOLOGY

27. Broadly speaking, there are two types of epidemiologic studies: descriptive and analytic. Descriptive studies, which are most often conducted on data from disease and mortality registries maintained by public health departments and government agencies, are generally used as surveillance tools to monitor the temporal trends and spatial distribution

[5] Technically, distinction should be made between exposure, which refers to the concentration of the agent of interest in the ambient environment, and dose, which refers to the concentration of the agent in the biologic tissue of interest. However, in epidemiology, this distinction is not often made because most epidemiologic studies deal with exposures in the ambient environment. In this report, exposure and dose will be used interchangeably.

of diseases. Such studies can raise concern if either the temporal trends or spatial distribution of the disease under consideration show unusual or unexpected patterns but cannot establish causal associations between a suspect environmental factor and the disease of interest. However, carefully collected registry data[6] can form the basis of analytic studies that shed light on factors influencing temporal trends in disease and, in the case of cancer registry data, provide insights into mechanisms of carcinogenesis (e.g., Armitage and Doll, 1954; Moolgavkar and Knudson, 1981; Luebeck and Moolgavkar, 2002; Jeon et al., 2006; Meza et al., 2008).

28. The most common analytic epidemiology studies used to investigate associations between exposures and disease are the cohort and case-control studies. When a number of such independently conducted analytic studies yield consistent results, epidemiologists conclude that the association between the exposure and the disease could be causal. In general, however, to infer causality, in addition to the finding of an association, epidemiologists consider guidelines often referred to as the Hill criteria, after Sir Austin Bradford Hill, a British statistician who first enunciated them (Hill, 1965). If, on the other hand, repeated analytic studies find no evidence of an association between the exposure and the disease, it is safe to conclude that the exposure does not increase the risk of the disease, and is, therefore, not causally associated with the disease. These principles are generally accepted in the scientific community.

Measures of risk

29. The two main kinds of analytic studies in epidemiology are cohort and case-control studies.

    a. In a cohort study, the incidence of the disease under investigation is compared among groups of individuals who are exposed and unexposed to the environmental agent of interest.
    b. In a case-control study, on the other hand, exposures to the agent of interest are compared between diseased and non-diseased individuals.

30. The target of estimation in both cohort and case-control studies is the relative risk (RR); *i.e.*, the risk of disease among exposed individuals divided by the risk of disease among unexposed individuals. An RR of 1 or smaller indicates no increased risk associated with exposure. As with all statistical procedures, there is a margin of error associated with the estimation of RR from epidemiological studies. This margin of error is usually reported as a 95% confidence interval (CI), which is a range of values containing the estimated RR. If the 95% CI includes 1, the estimated RR is statistically insignificant; if the 95% CI does not include 1, the RR is statistically significant. A statistically significant RR greater than 1 suggests that exposure may be causally associated with the disease. However, in epidemiological studies, one statistically significant result suggests only an association. Inferences regarding causality are strong when multiple analytical studies in different populations all yield similar results.

[6] Population-based cancer registries are also often used for identification of cases for case-control studies.

EPIDEMIOLOGY OF MESOTHELIOMA

31. Mesothelioma has often been called a sentinel or signal tumor for asbestos exposure. Plaintiffs' experts, including Dr. Brodkin,[7] often misinterpret this statement to mean that mesothelioma cannot occur without exposure to asbestos. In fact, the correct interpretation of this statement is that every case of mesothelioma should arouse suspicion of possible asbestos exposure. There is a fundamental misperception that every case of mesothelioma involves exposure to asbestos. In fact, like every other cancer, mesothelioma can occur spontaneously, without exposure to any external agent, as discussed below. Moreover, in addition to exposure to amphibole asbestos, exposure to ionizing radiation can also increase the risk of mesothelioma (Travis et al., 2005; Tward et al., 2006; Teta et al., 2007; Hodgson et al., 2007; De Bruin et al., 2009; Goodman et al., 2009; Gibb et al., 2013; Farioli et al., 2013).

Epidemiologic studies of asbestos and mesothelioma

32. A number of analytical epidemiology studies have investigated the association between exposure to asbestos and the occurrence of mesothelioma. These studies have shown that occupational exposure to amphibole asbestos is causally associated with mesothelioma. However, these studies have also shown unequivocally that all occupational exposures are not equal in their potential to cause mesothelioma. To quote Irving Selikoff et al. (1965), one of the pioneers in the study of asbestos and mesothelioma, "It is inadequate to speak now of 'asbestos workers'. With the growth of asbestos utilization, including rapid multiplication of the number and variety of its applications, it would perhaps be more accurate to categorize workmen exposed to asbestos as 'asbestos textile workers', 'asbestos insulation workers', 'asbestos miners', 'asbestos mill workers', 'asbestos-cement workers', etc. **The different occupations vary widely in important respects; in intimacy, intensity and duration of exposure, in variety and grade of asbestos used, in working conditions, in concomitant exposure to other dusts or inhalants."** (emphasis added). Thus, there is no scientific support for the position that all asbestos exposure contributes equally to the development of mesothelioma.

Mesothelioma can occur without exposure to asbestos

33. In addition to the general biological argument, several lines of evidence support the conclusion that mesothelioma can occur without asbestos exposure. Mesothelioma is known to occur in individuals with no history of exposure to asbestos. Epidemiologic studies (*e.g.*, Pelnar, 1988; Peterson et al., 1984; Spirtas et al., 1994; Agudo et al., 2000; Teschke et al., 1997; Rake et al., 2009) report that a significant fraction of mesothelioma cases had no history of exposure to asbestos, with a larger fraction of female cases

[7] When Dr. Brodkin was asked in his deposition whether he found any evidence of other asbestos-related conditions in Mr. Quirin, he responded, "Certainly the malignant mesothelioma is a signal marker for asbestos exposure, but in terms of nonmalignant markers, I didn't see any evidence of plaques on the contralateral right side or evidence of asbestosis."

reporting no exposure. Spirtas et al. (1994) reviewed the literature and reported that estimates of the fraction of mesothelioma cases exposed to asbestos in epidemiologic studies have varied from a low of 13% to a high of 100%.

34. Spirtas et al. (1994) undertook a formal analysis to estimate the fraction of mesothelioma cases that could be attributed to asbestos exposure. They concluded that, in their study, among men, 88% of pleural mesotheliomas and 58% of peritoneal mesotheliomas could be attributed to asbestos exposure. Among women, they could not estimate the population attributable fraction (PAF) separately for pleural and peritoneal mesotheliomas, but reported that only 23% of all mesotheliomas were attributable to asbestos exposure. This study provides strong evidence that both pleural and peritoneal mesothelioma can occur without asbestos exposure and that the fraction of peritoneal mesothelioma occurring without asbestos exposure is larger than the fraction of pleural mesothelioma occurring without asbestos exposure.

35. A recent large case-control study of mesothelioma in Great Britain (Rake et al., 2009) concludes that 86% of male and 38% of female cases were attributable to either occupational or domestic asbestos exposure. Price and Ware (2009) estimate that approximately 70–75% of mesotheliomas among males and only 3–10% of mesotheliomas among females in the U.S. in 2008 were attributable to asbestos exposure. Aguilar-Madrid et al. (2010) conclude that only 44% of cases of pleural mesothelioma in Mexico over the period 2004–2006 were attributable to occupational asbestos exposure. Lacourt et al. (2010) show that control selection can have a substantial impact on estimates of PAF from case-control studies, but conclude that the PAF for mesothelioma, i.e., the fraction of cases attributable to asbestos exposure, in France was between about 50% and 70%. Mesothelioma has also been reported to occur in young children and even congenitally (World Trade Organization [WTO], 2000; Huncharek, 2002). Such cases are manifestly not associated with asbestos exposure. Thus, mesothelioma can clearly occur idiopathically.

36. A recent analysis of the Surveillance, Epidemiology and End Results (SEER) data (Price and Ware, 2004) indicates that the background (spontaneous) rate of mesothelioma (pleural and peritoneal combined) in the United States is between 2 and 4 cases per million individuals per year and that the lifetime risk of spontaneous mesothelioma is between 2 and 4 per 10,000 individuals. Price and Ware (2004) do not provide separate estimates for pleural and peritoneal mesothelioma.

37. In an updated analysis of the SEER data, Moolgavkar et al. (2009) estimate that the background rate of peritoneal mesothelioma in the U.S. is approximately 1 per million per year and that the lifetime risk of spontaneously occurring peritoneal mesothelioma is approximately 1 in 10,000. For pleural mesothelioma, Moolgavkar et al. (2009) estimate that background rates are between 2 and 3 per million individuals per year and the lifetime probability is approximately 3 per 10,000 individuals. A recent large case-control study of mesothelioma (Rake et al., 2009) in the U.K. estimated a lifetime risk of mesothelioma (pleural and peritoneal combined) among individuals not exposed to asbestos of about 1 in 1,000, approximately three times that estimated by Moolgavkar et

al. (2009) in the U.S.

38. Observed trends in mesothelioma incidence in the SEER registry over the period 1973 to 2005 also strongly support the notion that some fraction of mesotheliomas over this period of time was not attributable to asbestos exposure. Figure 1 shows the trends in age-adjusted rates of mesothelioma over this period of time.

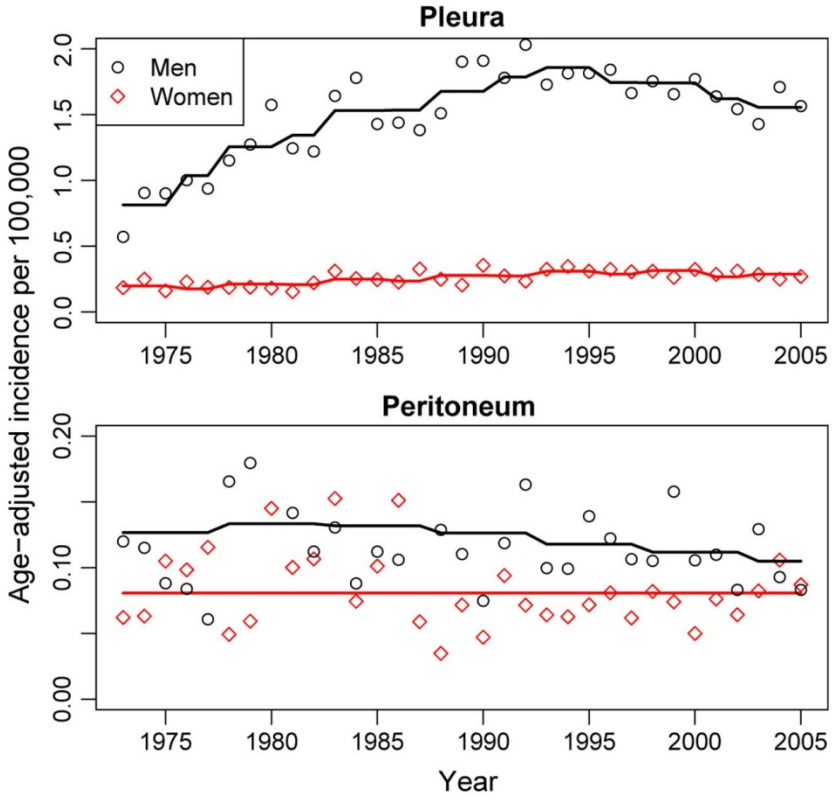

Figure 1. *Observed and expected age-adjusted incidence rates over the period 1973–2005. Upper panel: pleural mesothelioma; lower panel: peritoneal mesothelioma (from Moolgavkar et al., 2009).*

39. Age-adjusted incidence of pleural mesothelioma among males increased from 1973 to the early nineties and appears to be declining thereafter. This trend reflects the trend in the commercial use of asbestos in the U.S. with an approximately forty year lag. The incidence rates of pleural mesothelioma among females shows a much weaker trend suggesting that a large fraction of pleural mesotheliomas among females over this period cannot be attributed to asbestos exposure. The age-adjusted incidence of peritoneal mesotheliomas in both males and females is essentially flat. The lack of trends in incidence reflecting trends in asbestos usage suggests that a large fraction of peritoneal mesothelioma in both males and females over the period 1973 to 2005 cannot be attributed to asbestos exposure.

40. Trends in some European countries also indicate that a large proportion of peritoneal

mesotheliomas in those countries is unrelated to asbestos exposure. Hemminki and Li (2003) examined trends in the incidence of peritoneal mesothelioma in Sweden over the period 1961 to 1998. Among men, they reported that only 29% had "typical asbestos-related jobs …." Interestingly, the age-adjusted incidence rates were virtually identical in men and women. Since men were much more likely to be occupationally exposed to asbestos, this finding suggests that a large fraction of peritoneal mesotheliomas in Sweden over this period were unrelated to asbestos exposure. Moreover, the generally increasing trends in incidence over this period are probably attributable to factors other than asbestos.

41. Burdorf et al. (2007) examined the incidence of peritoneal mesothelioma among men and women in Sweden and the Netherlands over the period 1989 to 2003, and reported that the trends were flat. They concluded, "[t]he absence of a time trend in the incidence rate of peritoneal mesothelioma in Sweden and the Netherlands in the past 15 years may point to a more limited role of occupational exposure to asbestos in the aetiology of peritoneal mesothelioma than for pleural mesothelioma, especially among women."

42. The discussion of peritoneal mesothelioma is relevant here because, even though Mr. Quirin suffered from pleural mesothelioma, it underscores the fact that mesothelioma can occur in the absence of exposure to asbestos.

Chrysotile and mesothelioma

43. Plaintiffs' experts are often opine that pure chrysotile, uncontaminated by amphiboles, can cause mesothelioma. The issue is far from settled, however, and continues to be debated in the literature (e.g., Yarborough, 2006, 2007).

44. The only way to determine whether pure chrysotile can increase the risk of mesothelioma is to conduct properly designed epidemiologic studies. But herein lies the problem: for most epidemiologic studies of mesothelioma among workers supposedly exposed to pure chrysotile, it has later been reported that the chrysotile was, in fact, contaminated by amphiboles. For example, Dr. Brodkin cites the study by Yano et al. (2001) in China reporting mesothelioma in a factory using chrysotile to support his contention that pure chrysotile can cause mesothelioma; however, Chinese chrysotile has been shown to be contaminated by amphiboles (Tossavainen et al., 2001). Furthermore, it is now known that one of the two cases of mesothelioma in the Yano study was probably exposed to high levels of asbestos (of unknown type) as a result of a textile weaving business run by his parents in their home (Yano et al., 2009). Yano et al. (2009) also performed a lung burden analysis on one of the cases of mesothelioma and found that the main fiber type in the lung was tremolite, an amphibole. Thus, these cases were clearly exposed to substantial quantities of amphibole asbestos.

45. Dr. Brodkin also cites the studies of Italian chrysotile miners in Balangero, Italy (Mirabelli et al., 2008; Pira et al., 2009). He fails to note, however, that these miners were exposed to the fiber balangeroite, in addition to chrysotile. There is evidence that the toxicological properties of balangeroite are similar to those of amphibole asbestos

(Groppo et al., 2005; Turci et al., 2005; Gazzano et al., 2005). Furthermore, some crocidolite,[8] the most potent form of amphibole asbestos, was processed at Balangero (Browne, 2001). A recent paper by Turci et al. (2009) suggests that balangeroite may not possess the toxic properties of amphiboles; however, this suggestion is based purely on *in vitro* studies, rather than epidemiologic studies. Epidemiologic studies on balangeroite would be impossible to conduct because the mineral is present only in combination with chrysotile. At the very least, properly designed *in vivo* studies are required to address the issue of whether the presence of balangeroite contributed to the risk of mesothelioma at Balangero. While Dr. Brodkin relies on the Yano and the Balangero studies, he fails to note that these studies of workers exposed to large quantities of chrysotile asbestos contaminated with amphibole or amphibole-like substances have little relevance to the risks associated with exposure to much lower levels of amphibole-free chrysotile.

46. Some plaintiffs' experts use an argument advanced by Smith and Wright (1996) to contend that chrysotile asbestos is the predominant cause of pleural mesothelioma. This argument has been convincingly countered by Hodgson and Darnton (2000) who state, "What this argument ignores is any quantification of exposure. Without quantification it is very difficult to draw any conclusion about relative risk from a simple ranking by mesothelioma rate. In relation to the 25 cohorts identified in this review an equally pertinent observation might be that all of them involved exposure to one or other of the amphibole fibres."

47. Kanarek (2011) reviewed the literature on exposure to chrysotile and mesothelioma and concluded that chrysotile alone, uncontaminated with amphibole, can cause mesothelioma. Yarborough (2006, 2007) reviewed the same epidemiologic studies reviewed by Kanarek (2011) except for the few that have appeared after 2006 and arrived at a completely different conclusion. Yarborough noted that contamination with, or use of, amphibole occurred in most so-called 'pure' chrysotile cohorts. I have reviewed in some detail here many of the recent studies that Kanarek (2011) relies on for his conclusions. Kanarek (2011) relies on a mix of case reports and epidemiologic studies and it is clear that he has not critically reviewed the literature that he cites. An egregious example is provided by his reliance on the study by Madkour et al. (2009), a study on which Dr. Brodkin also relies. This is a study that reports high risks associated with environmental exposure to asbestos around a cement plant in Egypt; however, the results reported in the paper suggest strongly that environmental and occupational exposures may not have been accurately estimated. For example, Table 1 of Madkour et al. (2009) reports that mesothelioma risks are far higher in those environmentally exposed than in those occupationally exposed (who are also presumably environmentally exposed if they live in the vicinity of the plant). About the only conclusion that can be drawn from the study is that the rates of mesothelioma are high in the vicinity of the cement plant. Moreover, several publications (Emara et al., 1970; Kamal et al, 1992; Gaafar and Eldin, 2005; Gaafar, 2007) report the presence of crocidolite at Egyptian cement factories. Another major deficiency of Kanarek's paper is that he relies on case reports for his

[8] Pooley (unpublished, 1990) has reported the presence of amphiboles, including crocidolite, in the lung tissue of workers at the Balangero mines and residents of the area.

conclusions even when multiple well-conducted epidemiologic studies arrive at completely different conclusions from those he arrives at based on case reports. An example is provided by his discussion of brake repair workers in the U.S. I consider this in more detail below.

48. While there is still debate as to whether or not pure chrysotile increases the risk of mesothelioma, there is little doubt that amphiboles are much more potent mesotheliogens than chrysotile, if chrysotile causes mesothelioma at all. The epidemiologic evidence, discussed and summarized both by Hodgson and Darnton (2000) and Berman and Crump (2003; 2008a, b), demonstrates convincingly that exposure to amphiboles is far more potent than exposure to chrysotile as a cause of mesothelioma. Hodgson and Darnton (2000) concluded that amosite is 100 times and crocidolite 500 times as potent as chrysotile in causing mesothelioma. Berman and Crump (2008b) considered various measures of exposure as they relate to the risk of mesothelioma in 11 cohorts occupationally exposed to asbestos. They concluded that the hypothesis that chrysotile and amphibole asbestos are equally toxic was strongly rejected no matter which measure of exposure was used. The estimates of the potency of chrysotile relative to that of amphibole ranged from zero to about 0.005, depending on the measure of exposure used. Epidemiologic studies of vehicle mechanics exposed to low levels of chrysotile asbestos have failed to reveal an increased risk of mesothelioma, as discussed below.

49. Dr. Brodkin completely ignores these studies, which are the only systematic attempts at evaluating, in a statistically rigorous way, the relative potencies of amphiboles and chrysotile. Instead, in his deposition, he opines that the mesotheliogenic potency of amphiboles is about three times that of chrysotile. He provides no support for his opinion. Plaintiffs' experts often opine that the estimates of relative potency derived by Hodgson and Darnton (2000) and Berman and Crump (2003) cannot be considered reliable because of serious problems with exposure assessment. In fact, these authors readily acknowledge that there are problems with accurate assessment of exposure to exposure. Hodgson and Darnton (2000) choose to consider only the mean cumulative exposure in each cohort because they argue that this measure is less prone to error than individual-level estimates of exposure. Berman and Crump (2003) use methodology that is completely different from that used by Hodgson and Darnton (2000). Whereas Hodgson and Darnton (2000) draw their conclusions regarding relative potency by looking at the fraction of mesothelioma deaths in an occupational cohort as a function of mean cumulative exposure in that cohort, Berman and Crump (2003) consider individual level exposures for each member of the cohort and model the risk of mesothelioma as a function of both intensity of exposure and duration of exposure using a model originally developed by Julian Peto and adopted by the EPA for its 1986 risk assessment (Nicholson, 1986). The fact that these two completely different methods of analyses yield broadly similar estimates of relative potency – amphiboles are approximately two orders of magnitude more potent than chrysotile – suggests strongly that the estimates are reliable.

50. Plaintiffs' experts also cite a recent analysis of a textile worker cohort in North Carolina (Loomis et al., 2009), which reported an increased risk of mesothelioma among workers

exposed predominantly to chrysotile asbestos. There were eight cases of pleural cancer, of which four were confirmed to be mesothelioma. Three of the cases of pleural cancer occurred in a plant where some amosite had been used; however, all confirmed cases of mesothelioma had no recorded exposure to amphiboles,[9] although most of the chrysotile used in the plants was Canadian and could have been contaminated with tremolite. The Hodgson-Darnton index for pleural cancer (including mesothelioma) in this study was reported to be 0.0058% per f/ml-year in the entire cohort and 0.0098% per f/ml-year among workers followed for at least 20 years. Based on the latter figure, Hodgson and Darnton (2010) suggested that the mesothelioma risk associated with exposure to chrysotile asbestos could be about an order of magnitude higher than estimated in their 2000 paper; however, this figure is an upper bound on risk because all pleural cancers, some of which may not have been mesotheliomas, were included in the calculation and the calculation was done only on workers who were followed for at least 20 years. Moreover, three of the eight pleural cancers occurred in a plant where amosite had been used. Based on the Loomis et al. (2009) paper, the relative potency of amphiboles to chrysotile may have to be revised down from the estimates in Hodgson and Darnton (2000) and Berman and Crump (2008a, b). It is clear, however, despite the new information in the Loomis et al. (2009) paper, that amphibole asbestos is considerably more potent than chrysotile as a mesotheliogen, if pure chrysotile causes mesothelioma at all.

51. If, on further review, it is confirmed that the cases of mesothelioma in the North Carolina cohort had not been exposed to amphiboles, then the relative potency of amphibole to chrysotile will have to be revised downward. There have been instances in the past, however, when on more careful examination, so-called pure chrysotile cohorts were discovered to have been exposed to amphiboles. For example, the South Carolina Textile Workers' cohort has sometimes been referred to as a pure chrysotile cohort. But, in fact, small quantities of crocidolite were used at this plant between the 1950s and 1975 (Yarborough, 2006, 2007). Moreover, lung burden analyses (Green et al., 1997; Case et al., 2000) indicated also that this cohort was exposed to commercial amphiboles. Another example is provided by the Yano et al. (2001) cohort discussed above. Although, as I have noted above, Dr. Brodkin relies on the Yano et al. (2001) paper, he does not acknowledge the facts that Chinese chrysotile contained tremolite (Tossavainen et al, 2001), that at least one of the two cases of mesothelioma in the Yano cohort had been heavily exposed as a child and that the predominant fiber type found in the lungs of one of the cases was tremolite (Yano, 2009).

52. Toxicological evidence also supports the conclusion that chrysotile has much lower carcinogenic potency than amphiboles. Recent work (e.g., Mossman et al., 2011) show that chrysotile is more rapidly cleared from tissues than amphibole asbestos suggesting that it can be more effectively dealt with by the defense mechanisms of the body and is

[9] As indicated elsewhere in this report, lung burden analyses have shown that members of cohorts thought to be exposed predominantly to chrysotile have amphiboles in their lungs (e.g., Green et al., 1997; Case et al., 2000). Interrogatories suggest that substantial quantities of amphibole could have been used at the North Carolina facility at issue when it was operated by Unarco Industries.

less biopersistent. Analyses of toxicological data on fiber biopersistence have shown that biopersistence is strongly correlated with carcinogenicity (Moolgavkar et al., 2001). Other experimental work (Shukla et al., 2004, 2009) shows that chrysotile is less potent than other types of asbestos in triggering critical biological events thought to be important in the carcinogenic process.

Well-conducted epidemiologic studies fail to report an increased risk of mesothelioma among vehicle mechanics suggesting that there is a level of exposure to chrysotile asbestos below which no increase in the risk of mesothelioma can be detected

53. The debate over whether pure chrysotile uncontaminated with amphiboles can increase the risk of mesothelioma is largely academic because most commercially available chrysotile has some amphibole contamination, primarily as tremolite. The important issue is whether exposure to low levels of commercial chrysotile, whether or not contaminated with amphibole, can increase the risk of mesothelioma. One way to address this question is to conduct properly designed epidemiologic studies of occupations exposed to low levels of chrysotile asbestos.

54. The issue of whether exposure to low levels of commercial chrysotile increases the risk of mesothelioma can be addressed in epidemiologic studies of the association between work as an automobile mechanic and mesothelioma. Because brake pads contain chrysotile asbestos bound in a resin, vehicle mechanics are potentially exposed to it when engaged in work on brakes.[10] Garage and brake mechanics have, therefore, been included in a number of analytic epidemiologic studies investigating the risk of mesothelioma in specific occupational groups. These studies have been conducted by independent and reputable researchers.

55. The most important of these studies are 13 case-control studies (McDonald and McDonald, 1980; Teta et al., 1983; Spirtas et al., 1985, 1994; Woitowitz and Rödelsperger, 1994; Teschke et al., 1997; Agudo et al., 2000; Hansen and Meersohn, 2003; Hessel et al., 2004; Welch et al., 2005; Rolland et al., 2005, 2010; Rake et al., 2009; Aguilar-Madrid et al., 2010), all of which conclude that vehicle mechanics are not at increased risk of mesothelioma. In fact, the study by Teschke et al. (1997) concludes that vehicle mechanics are at no greater risk of mesothelioma than accountants and school teachers. These studies reported estimated RRs associated with work as a motor vehicle mechanic or brake worker fluctuating around 1, with most RRs being actually less than 1.0 and not a single RR being statistically significant (Table 1). Moreover, Hessel et al. (2004) and Peto et al. (2009), which is the full report on which Rake et al. (2009) is based, find no evidence that work as a vehicle mechanic increases the risk of mesothelioma associated with other asbestos exposures.

56. In addition to the 13 case-control studies cited above, four cohort studies of vehicle mechanics in Europe (Hansen, 1989; Järvholm and Brisman, 1988; Gustavsson et al.,

[10] The time-weighted average (TWA) for exposure to chrysotile fibers longer than 5 μm over a work day for brake mechanics has been estimated to be 0.04 f/ml (Paustenbach et al., 2003), and the upper bound on cumulative exposure is approximately 3 f/ml-year (Finley et al., 2007).

1990; Merlo et al., 2010) likewise provide no evidence of an association between work as a vehicle mechanic and mesothelioma.

Table 1. *Epidemiologic studies of automobile mechanics and mesothelioma*

Author	*Year*	*Increased Risk (Yes / No)?*
Case Control Studies		
McDonald and McDonald	1980	No
Teta et al.	1983	No
Spirtas et al.	1985	No
Woitowitz and Rödelsperger	1994	No
Teschke et al.	1997	No
Agudo et al.	2000	No
Hansen and Meersohn	2003	No
Hessel et al	2004	No
Rolland et al.	2005, 2010	No
Welch et al.	2005	No
Rake et al.	2009	No
Aguilar-Madrid et al.	2010	No
Cohort Studies		
Järvholm and Brisman	1988	No
Hansen	1989	No
Gustavsson et al.	1990	No
Merlo et al.	2010	No
PMR Studies		
Petersen and Milham	1980	No
Olsen and Jensen	1987	No
Coggon et al.	1995	No
Hodgson et al.	1997	No
Milham and Ossiander (WOMD)	2001, 2011	No
NIOSH	2002	No
McElvenny et al.	2005	No
Roelofs et al.	2013	Yes

57. Similarly, seven descriptive studies, the so-called proportional mortality ratio (PMR) and proportional incidence ratio (PIR) studies (Petersen and Milham, 1980; Olsen and Jensen, 1987; Coggon et al., 1995; Hodgson et al., 1997; Milham and Ossiander, 2001;[11] National Institute for Occupational Safety and Health [NIOSH], 2002; McElvenny et al. 2005), provide no evidence of an association between work as a vehicle mechanic and mesothelioma. These studies indicate that the fraction of deaths due to mesothelioma or

[11] Milham and Ossiander (2001) is an unpublished government report discussing various results and analyses from the Washington Occupational Mortality Database (WOMD). These data were updated in 2011. The updated data and analyses from the WOMD are available on the following website: https://fortress.wa.gov/doh/occmort/OMQuery.aspx (Washington Department of Health, 2011).

the fraction of disease attributable to mesothelioma among vehicle mechanics is not different from other occupational groups in which exposure to asbestos does not occur.

58. A recent PIR study (Roelefs et al., 2013) reported a statistically significant increased standardized incidence odds ratio (SIOR) among automobile mechanics. The SIOR was estimated by exploiting a formal equivalence between the standardized incidence ratio (SIR) design and the case-control design. Despite this formal equivalence, the SIOR derived from this PIR study is not a reliable indicator of an increased risk of mesothelioma among automobile mechanics. Like any PIR study, this study has information on only a single occupation for each subject in the study making the control of confounding impossible. A second serious problem with this study is the biased selection of controls. However controls are chosen, their occupations must be representative of the occupations in the population in which the mesotheliomas arise (i.e., the base population). Life-style habits, such as smoking, that influence the risk of many cancers are different in the different occupations. Occupations, such as work as an automobile mechanic, in which smoking rates are typically high (Bang and Kim, 2001; Leigh, 1996) will have a higher incidence and prevalence of the smoking-associated cancers. By eliminating these cancers from the comparison group, the authors have selected out a proportion of individuals in some occupations and, therefore, inflated the SIOR for such occupations, including automobile mechanics. In other words, by removing the lung cancers as possible comparison cancers, the authors have decreased the probability of selecting comparison cancer patients employed in occupations with a high prevalence of smoking, and therefore increased the SIOR for these occupations.

59. When the entire body of epidemiologic literature is evaluated, it is absolutely clear that work as an automobile mechanic does not increase the risk of mesothelioma. A single study using the weakest of epidemiologic designs and with other serious methodologic flaws cannot change that conclusion.

60. Kanarek (2011) does not mention any of these multiple epidemiologic studies exonerating exposure to low-level chrysotile asbestos as a cause of mesothelioma. He relies, instead, on a series of case reports and studies based on case reports to support his conclusion that brake repair work increases the risk of developing mesothelioma. Dr. Brodkin, who relies on the Kanarek study, does not allude to this serious limitation of the study. Kanarek's conclusion is as untenable as his methods for arriving at it. In fact, Nicholson et al. (1984) estimated that there were approximately 5,000,000 million current and former automobile mechanics in the U.S. at the time of his study. Based on a background rate of 3 to 4 per million person-years for idiopathic mesothelioma, one would expect 300 to 400 cases of mesotheliomas to arise spontaneously among automobile mechanics over a 20-year period.

61. The few available epidemiologic studies of mesothelioma among workers potentially exposed to joint compound have been recently reviewed by McCoy et al. (2010). These studies provide no evidence that exposure to joint compound increases the risk of mesothelioma. This finding is consistent with the conclusion derived from the epidemiologic studies of automobile mechanics that exposure to low levels of chrysotile

asbestos does not increase the risk of mesothelioma. The conclusions in McCoy et al. (2010) have been criticized by Dement and Lipscomb (2012). I agree that the McCoy et al. (2010) paper does not provide affirmative evidence that exposure to joint compound does not increase the risk of mesothelioma; however the McCoy paper does show that there is no epidemiologic evidence of an increased risk of mesothelioma from exposure to joint compound. Phelka and Finley (2011) reviewed the health hazards associated with exposures to asbestos-containing drywall-accessory products and concluded that there was no evidence of increased risk of mesothelioma. These studies are not cited by Dr. Brodkin and apparently were not considered by him in arriving at his opinions. Dr. Brodkin was asked about the McCoy paper at his deposition and appeared to suggest that the data in the paper actually supported an increased risk of mesothelioma from exposure to joint compound. This conclusion is totally untenable scientifically.[12]

62. In addition to these published studies, I have personally performed analyses of mesothelioma rates in the SEER data for San Benito and Monterey Counties, where UCC's Calidria mines are located (Moolgavkar, 2012). My analyses indicated that the mesothelioma incidence rates in San Benito and Monterey counties are not elevated when compared with rates in other California registries or with rates in the entire SEER registry. This finding indicates that persons living in the vicinity of the Calidria mines in California are not at increased risk of developing mesothelioma.

Epidemiologists do not arrive at conclusions on the basis of one or a few studies

63. Plaintiffs' experts often assert that the case-control studies of work as a vehicle mechanic and mesothelioma have limitations and are therefore unreliable. The specific criticisms they level at the case-control studies of mesothelioma are well known to epidemiologists. I recognize that epidemiologic studies have limitations because most epidemiologic studies rely on observational data, unlike randomized studies where investigators can assign exposures, such as experimental studies among animals and clinical trials for new medical therapies. Such assignment of exposure is not generally possible in epidemiologic studies of human populations because of ethical considerations. Therefore, to account for these potential limitations, epidemiologists do not arrive at conclusions on the basis of one or a few studies, relying instead on a larger body of evidence from which to draw conclusions. However, when multiple epidemiologic studies conducted by different investigators in different parts of the world yield consistent

[12] Dr. Brodkin argued that in the CARET study, one case of mesothelioma was found among plasterers, an occupational group that includes individuals working with joint compound. He failed to mention, however, that plasterers also included individuals who worked with spray-on insulation. Nothing is known about other asbestos exposure that this single case may have received. Dr. Brodkin also disputes McCoy's interpretation of a study performed by Stern et al. (2001) in which they report a non-significant PMR for mesothelioma among plasterers. Here again, Dr. Brodkin fails to acknowledge that plasterers could have been exposed to substantial quantities of amphibole asbestos. Dr. Brodkin points to the fact that on a detailed review of death certificates, 40 cases of mesothelioma were found among the plasterers in this study. He ignores, however, one of the fundamental tenets of epidemiologic investigation: the same methods must be applied to all groups in a study. The death certificates of all individuals in the study should have received the SAME scrutiny as the death certificates of the plasterers.

results, the findings can be considered reliable. This is exactly the situation with the epidemiologic studies of mesothelioma and work as a vehicle mechanic. Furthermore, these same studies have identified occupations, such as shipyard, insulation and construction work, causally associated with risk of mesothelioma.

Evidence cited by plaintiffs' experts in support of the proposition that low exposures to asbestos can increase the risk of mesothelioma is fatally flawed

64. Plaintiffs' experts often rely on case-control studies conducted by Iwatsubo et al. (1998) and Rödelsperger et al. (2001) and Lacourt et al. (2010) to conclude that even very small exposures to asbestos can increase the risk of mesothelioma substantially; however, a critical review of these studies reveals that, in addition to the usual limitations of epidemiologic studies, these papers contain fatal flaws, which makes their quantitative conclusions unreliable. Moreover, the results reported in these studies are inconsistent with each other and with other epidemiologic literature on exposure-response relationships in mesothelioma.

65. Iwatsubo et al. (1998) conducted a case-control study of pleural mesothelioma in France to examine exposure-response relationships at low exposure levels. They had no direct information on actual exposure levels in the various jobs held by the subjects of the study. The authors had a panel of industrial hygienists reconstruct exposure, although there is only sparse description of how this was done and no references to typical exposure levels in the various job categories are provided. For the latter point, due to the latency period for mesothelioma, the authors were interested in exposure levels that existed 20 or more years prior to the diagnosis; however, these exposure levels are generally unknown or highly uncertain. It is highly likely that Iwatsubo et al. (1998) under-estimated exposure substantially, particularly at the low exposure levels. First, they defined low exposure intensity as being less than 1 f/ml, but in their exposure reconstruction, they assigned an exposure concentration of 0.1 f/ml to the low exposure category. Similarly, they define medium exposure intensity as being between 1 and 2 f/ml, but assign a concentration of 1 f/ml to exposures in this category in the reconstruction process. Thus, they could have under-estimated exposure by a factor of 10 in the lowest exposure intensity group and by a factor of 2 in the medium intensity exposure group. Second, they did not consider any exposure during the 20 years prior to the diagnosis of mesothelioma (under the assumption that the latency period for mesothelioma is at least 20 years). The EPA model for mesothelioma (Nicholson, 1986; Berman and Crump, 2003; 2008a) assumes that the latency period is 10 years. By ignoring exposures that occurred between 10 and 20 years before the diagnosis of the disease, the investigators could have substantially under-estimated total exposures. As a consequence of the under-estimation of exposure, the investigators have in all probability grossly over-estimated the mesothelioma risks associated with low cumulative exposure to asbestos.[13]

[13] The fact that these authors obtained a positive exposure-response relationship (*i.e.*, higher risks were associated with higher exposures) in this study suggests that the rank ordering of the exposures was correct, even if the absolute exposure levels were incorrect.

66. There are other problems with this study, including:

    a. The investigators could not distinguish between asbestos fiber types, so that their conclusions cannot be applied to exposure to pure chrysotile.

    b. The investigators designed their study as a matched pairs study; however, because they could not get matched controls for all the cases, they broke the matching for their analyses. This procedure could have led to substantial bias in their estimates of risk, although the direction of the bias cannot be predicted.

    c. The investigators report that there were more blue-collar workers among cases than controls. This could bias the estimates of risk upwards since blue collar workers are more likely to be exposed to asbestos in the workplace.

67. Rödelsperger et al. (2001) conducted a case-control study of mesothelioma in Germany. As in the Iwatsubo et al. (1998) study, they had no direct information on asbestos exposure concentrations and had industrial hygienists estimate exposures associated with various job categories. Therefore, this study shares some of the serious limitations of the Iwatsubo study outlined above. Other specific limitations of this study include:

    a. As in the Iwatsubo study, the investigators could not distinguish between fiber type, though they recognized the importance of this distinction stating, "In addition, the type of asbestos – chrysotile or amphibole – is unknown in spite of its well-known importance."

    b. Lung fiber burden analysis in this study shows only modest correlation between exposure estimates and lung burden for amphiboles but no correlation for chrysotile, suggesting that the industrial hygiene exposure estimates could be in error.

    c. The reported results are highly sensitive to choices of cut-points for categorical exposures. Results are also sensitive to whether hospital-based or population-based controls are used.

68. Lacourt et al. (2010), reports on two case-control studies conducted in France using methods similar to those used by Iwatsubo et al. (1998) and suffers from the same deficiencies. In particular, the results of the two case control studies reported in the paper are not consistent with each other.

69. These three studies of low exposure levels thus share some of the same limitations. Furthermore, the results of the three studies are not consistent with each other, with the Rödelsperger study reporting much higher risks than the Iwatsubo and Lacourt studies. For example, the Rödelsperger study reports an odds ratio (OR) (a measure of relative risk) of 9.2 in the group exposed to less than 0.15 f/ml-years, whereas Iwatsubo et al. (1998) report an odds ratio of 8.7 in the group exposed to more than 10 f/ml-years.[14] In

[14] Both odds ratios assume a 20-year latency.

every exposure category, Rödelsperger et al. (2001) report risks that are far larger than the risks reported in Iwatsubo et al. or in Lacourt et al. (2010). These huge discrepancies in risk estimates among the three studies cannot be attributed simply to chance. They suggest fundamental problems with the exposure estimates in these studies. It is interesting to note in this regard that the highest estimated exposure concentration in the Iwatsubo study was 100 f/ml, whereas in the Rödelsperger study it was 10 f/ml, even though the studies covered approximately the same period of time. In the Lacourt study the highest exposure concentration was simply reported as being greater than 10 f/ml with no specific numerical value.

70. A recent analysis (Larson et al., 2010) of the cohort of miners at Libby, Montana, who were exposed to a mixture of amphiboles reports a significantly increased relative risk of 17.1 for mesothelioma only in the group exposed to more than 44 f/ml-year of the mixed amphibole fibers found at Libby. Thus, the risk of mesothelioma reported for the mixture of amphiboles found at Libby is far smaller than the risks reported in Iwatsubo et al. (1998), Rödelsperger et al. (2001), and Lacourt et al. (2010). Moolgavkar et al. (2010) estimated the potency of the Libby fibers for mesothelioma and concluded that it was in the middle of the range of potencies for the mixed fiber cohorts as reported in Hodgson and Darnton (2000) and Berman and Crump (2003; 2008a, b). Combined with the results reported by Larson et al. (2010), this finding strongly suggests that the estimates of risks in the Iwatsubo, Rödelsperger and Lacourt papers are much higher than, and inconsistent with, the risks in the mixed fiber cohorts considered by Hodgson and Darnton (2000) and Berman and Crump (2003; 2008a, b).

71. In a study of environmental and household exposures to crocidolite, the most carcinogenic form of asbestos, at Wittenoom, Western Australia, Reid et al. (2007) found that relative risks were below 10 even for cumulative exposures above 10 f/ml-year. Because of the high potency of crocidolite as a mesotheliogen, one would expect mixed fiber exposures, such as those investigated by Iwatsubo et al. (1998), Rödelsperger et al. (2001), and Lacourt et al. (2010) to carry lower risks than the Reid study of crocidolite exposures.

72. In conclusion, numerous well-designed epidemiologic studies of mesothelioma and work as a vehicle mechanic are consistent in suggesting that low levels of exposure to chrysotile asbestos do not pose an increased risk of mesothelioma. In contrast, three case-control studies (the Rödelsperger, Iwatsubo and Lacourt studies), the results of which are inconsistent with one another and with other literature on exposure-response relationships in mesothelioma, suggest that even low-level exposures to asbestos can increase the risk of mesothelioma. Given their serious deficiencies and inconsistent results, these latter studies cannot be considered to be reliable. Moreover, these studies involved mixed exposures to both chrysotile and amphiboles and therefore cannot address questions pertaining to low-level chrysotile-only exposures.

<u>Ionizing radiation can increase the risk of mesothelioma</u>

73. Plaintiffs' experts often do not acknowledge that asbestos is not the only cause of

mesothelioma in the U.S. As noted earlier in this report, recent epidemiologic literature provides strong evidence that ionizing radiation can cause mesothelioma (Travis et al., 2005; Tward et al., 2006; Teta et al., 2007; Hodgson et al., 2007; De Bruin et al., 2009; Goodman et al., 2009; Gibb et al., 2013; Farioli et al., 2013).

EXPOSURE-RESPONSE RELATIONSHIPS AND THRESHOLDS

74. Plaintiffs' experts argue that no threshold for asbestos-induced cancer has been demonstrated and, therefore, every exposure, no matter how small, is a substantial contributing factor to disease. The contention that there is no threshold for asbestos-associated cancer is based not on direct observation, but on mathematical risk assessments conducted by regulatory agencies. Risk assessment is a tool used to put bounds on the possible risks associated with specific exposure scenarios. In the absence of relevant data, a number of conservative assumptions are used in the process of risk assessment. By conservative, I mean the assumptions are chosen to err on the side of safety, i.e., they are much more likely to overstate, rather than understate, the risks associated with exposure. For all carcinogens, the EPA and other regulatory agencies start out with the **assumption** that there is no threshold. A quantitative risk assessment is not an epidemiological study and the results of the assessment should not be interpreted as meaning that a health problem has been detected at low exposure levels, which is what plaintiffs' experts contend.

75. A central problem in risk assessment is that most often potential risks have to be estimated at very low levels of exposure where no direct observations are available. In the case of asbestos, quantitative dose-response relations are based on occupational cohorts with average exposures greater than about 15 f/ml-year (Hodgson and Darnton, 2000). Risks at lower exposure levels are then estimated using a mathematical extrapolation, **which makes the explicit assumption that there is no threshold**. Moreover, it can be argued on theoretical statistical grounds that the existence of a threshold can never be established from epidemiological studies. However, the lack of evidence of a threshold is not equivalent to absence of a threshold. There may well be a threshold below which asbestos exposure causes no disease. We simply do not know whether or not a threshold exists. But, as I have discussed above, well-conducted epidemiologic studies have failed to demonstrate an increase in the risk of mesothelioma among vehicle mechanics who are exposed to low levels of chrysotile asbestos.

76. The problem of low-dose extrapolation, which is one of most contentious in quantitative risk assessment, is illustrated in Figure 2. In Figure 2, which diagrams the process of low dose extrapolation used in risk assessment, the observed data lie to the right of the dashed vertical line. The dose-response relationship in this region is depicted by a solid line. To the left of the vertical line, in the range of inference, no reliable information is available and the response (risk) in this region must be inferred from a mathematical relationship derived from observations in the observable range, *i.e.*, at high doses. The straight dashed line is based on the commonly used procedure of linear extrapolation.[15] This is

[15] However, see comments below regarding linearity of the mesothelioma models.

widely considered to be a conservative procedure in that it could, and probably does, over-estimate, but not under-estimate, the true risk. The possible true risks are shown by the curved dashed lines. The further one gets from the observable range, *i.e.*, the lower the dose, the less certain is the quantitative estimate of risk.

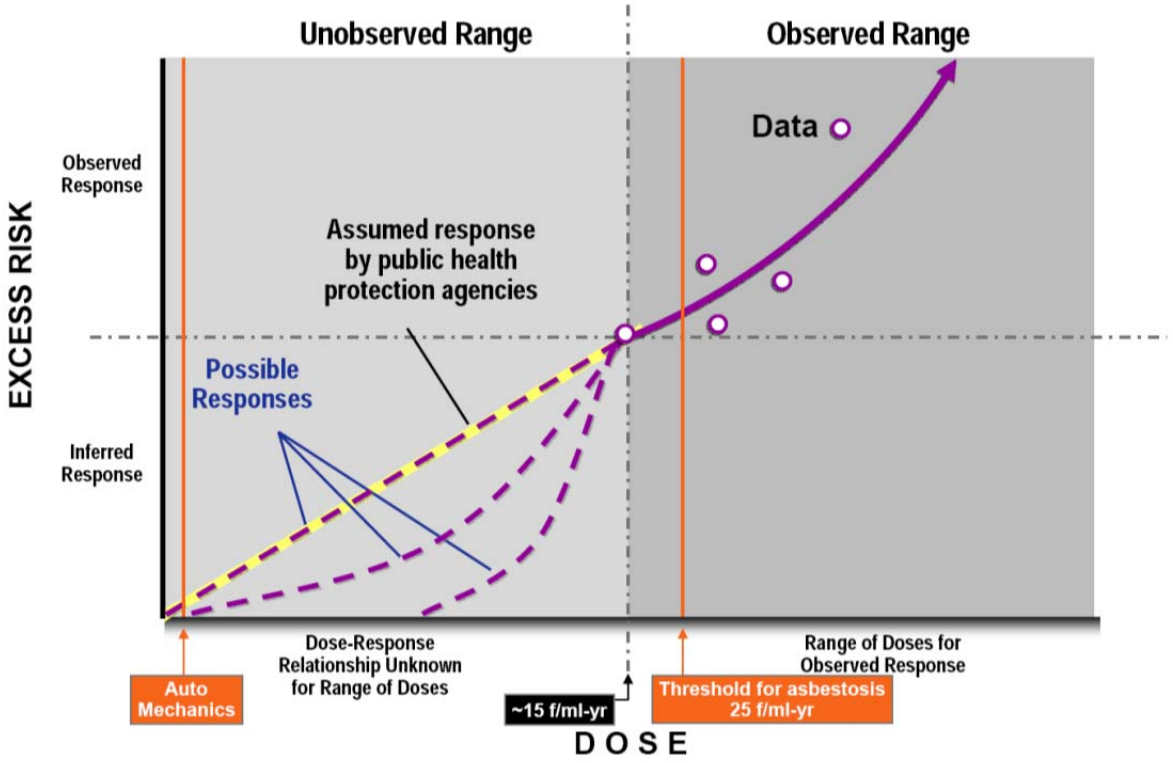

Figure 2. *The problem of low-dose extrapolation. The observed data lie to the right of the dashed vertical line in the zone of observation. Inferences regarding the risk (response) have to be made at much lower doses, in the zone of inference. Usually a mathematical dose-response function is fit to the data in the observed range and used to extrapolate risks to lower doses in the zone of inference. Agencies charged with protecting the public health often assume that the dose-response function is linear with no threshold. However, see comments below regarding linearity of the commonly used mesothelioma models. In fact, the actual shape of the dose-response curve in the zone of inference is unknown and could include a threshold. The figure shows also some critical benchmark exposure levels in asbestos epidemiology. Quantitative dose-response relationships in asbestos epidemiology are based on occupational cohorts with large cumulative exposures. The lowest average exposure among cohorts considered by Hodgson and Darnton (2000) is about 15 f/ml-year. The "threshold" for asbestosis is widely accepted to be 25 f/ml-yr (Doll and Peto, 1985). Finally, most career auto mechanics are exposed to about 3 f/ml-yr or less (Finley et al., 2007).*

77. Dose-response relationships for asbestos-associated cancer are based largely on occupational cohort studies with high levels of asbestos exposure; average exposures in these cohorts are generally greater than about 15 f/ml-year (Hodgson and Darnton, 2000). For asbestos, two distinct mathematical approaches can be used to extrapolate risks to low doses, one proposed by Hodgson and Darnton (2000), and another proposed by Peto

et al. (1982) and used by the EPA (Berman and Crump, 2003; ERG, 2003) and Berman and Crump (2008a, b). These two approaches yield distinctly different results showing that estimates of risk at low exposure levels are unreliable. In a recent discussion of issues in asbestos risk assessment, Case et al. (2011) concluded, "In summary, extrapolation from high to low risk, whether based on inferential statistical (e.g., linear no-threshold) models or mode-of-action-based models, is fraught with uncertainty."

78. Plaintiffs' experts often opine that the commonly-used models for asbestos risk assessment are linear. They are mistaken. In fact, neither the EPA model, based on the work done by Nicholson (1986), nor the Hodgson and Darnton (2000) model is linear in cumulative exposure. In the latter model (Hodgson and Darnton, 2000), which has separate expressions for pleural and peritoneal mesothelioma and by fiber type, the risk of mesothelioma is a non-linear function of cumulative exposure. The EPA model (Nicholson, 1986; Berman and Crump, 2008a, b) has separate terms for fiber concentration and duration of exposure. The mesothelioma risk is modeled as a linear function of fiber concentration and a power function of duration of exposure. As a consequence, in the EPA model, risk cannot be modeled as a function of cumulative exposure. For example, a cumulative exposure of 10 f/ml-year accrued as 2 f/ml over a five year period will carry a different risk from 10 f/ml-year accrued as 5 f/ml over a two year period. The EPA model only appears to be linear because the duration of exposure is kept constant for calculations involving the unit risk.

Not every exposure to asbestos is a significant contributing factor in the development of mesothelioma

79. From the discussion above, it is clear that there is absolutely no direct evidence that exposure to low levels of asbestos increases the risk of mesothelioma. The risks of mesothelioma at low levels of exposure are estimated by the use of mathematical extrapolations, and the results depend on which particular mathematical formula is used to make the extrapolation. Despite this fact, plaintiffs' experts use the "no threshold has been demonstrated" argument to contend that every exposure to asbestos, no matter how small, is a significant contributing factor to mesothelioma risk. When there are substantial other exposures to asbestos or radiation, plaintiffs' experts use a variation of this argument, the "last straw" argument, to contend that even the smallest exposure above the large exposure already experienced by the plaintiff from other sources increased substantially the risk of his/her mesothelioma because it was the last straw that broke the proverbial camel's back. The absurdity of the position that every exposure to asbestos makes a substantial contribution to the risk of disease in this situation can be seen from the following example. An individual who smoked two packs of cigarettes a day for forty years develops lung cancer and then claims that the second-hand smoke he breathed in as he walked past the open door of a bar was a substantial contributing factor in causing his disease.

80. Plaintiffs' experts ignore or dismiss the large and growing body of epidemiologic literature, consisting of multiple cohort, case-control, and PMR studies, which provides consistent and compelling evidence that work as an automobile mechanic, including work

on brakes, does not increase the risk of mesothelioma. They rely instead on a flawed syllogism. Their argument is that an increase in risk of mesothelioma is seen in occupational cohorts exposed to several hundred fiber/ml-yr of chrysotile contaminated with amphibole. Therefore, they argue, even an exposure two orders of magnitude below the exposure for which an increase in risk was observed must be associated with a substantial increase in risk under a non-threshold model.

81. Plaintiffs' experts, including Dr. Brodkin, often invoke the Helsinki Criteria to support their assertion that any history of exposure to asbestos in a case of mesothelioma is sufficient to conclude that the exposure caused the disease. This conclusion is simply wrong. The Helsinki Criteria (Henderson et al., 1997) are now more than ten years old and do not reflect the large amount of literature that has appeared since 1997. When the criteria were enunciated, it was widely believed that mesothelioma could not occur idiopathically and that asbestos was the only cause of mesothelioma. Both these beliefs are wrong as I have discussed above.

82.

a. First, there is compelling evidence that mesothelioma can occur spontaneously (idiopathically), that a substantial fraction of mesotheliomas, particularly peritoneal mesotheliomas occur idiopathically, and that as the use of asbestos declines, the fraction of idiopathic mesotheliomas is increasing.

b. Second, we know now that, in addition to amphibole asbestos, ionizing radiation can increase the risk of mesothelioma. To assert that any history of exposure to asbestos in a case of mesothelioma implies that the exposure caused the disease is completely analogous to saying that any history of exposure to cigarette smoke, no matter how small, in a lung cancer case implies that the exposure caused the disease. It is completely analogous to saying that any exposure to ionizing radiation such as a single CAT scan in a case of mesothelioma caused the mesothelioma. Plaintiffs' experts need to understand the concept of attributable or etiologic fraction, which is taught in any first-year course in epidemiology and is discussed below.

83. In fact, if Plaintiffs' experts want to make the argument that every exposure to asbestos over background is a substantial contributing factor to the development of mesothelioma, then they must be willing to accept that every exposure to ionizing radiation above background, including chest X-rays, is also a substantial contributing factor. Like asbestos, ionizing radiation is known to be a human carcinogen. Like asbestos, different forms of ionizing radiation have different carcinogenic toxicities. Like long-fiber amphibole asbestos, high-dose ionizing radiation has been shown to increase the risk of mesothelioma. Like asbestos, we are all exposed to a background level of ionizing radiation. And, like asbestos, there is considerable debate as to whether the dose-response relationship is monotonic without a threshold all the way down to the lowest doses. In fact, as for asbestos, regulatory agencies recognize no threshold for radiation-associated cancer.

84. Therefore, if Dr. Brodkin wants to assert that Mr. Quirin's bystander exposure to

chrysotile from joint compound was a substantial contributing factor to his mesothelioma, then he must be willing to also at least entertain the possibility that every chest X-ray that Mr. Quirin had and every airplane flight he took could also have contributed substantially to the risk. I see no evidence in Dr. Brodkin's notes that he even considered the possibility that ionizing radiation could have made a substantial contribution to the risk of Mr. Quirin's mesothelioma. The background dose of naturally occurring radiation is approximately 3 mSv/year.[16] A single round-trip flight[17] between New York and London exposes passengers, on average, to 0.1 mSv over background of ionizing radiation (Brenner et al., 2003). Thus, one round-trip flight between New York and London leads to incremental exposure equivalent to about 3.3% of the background radiation received naturally in one year.

85. The ATSDR (2001) estimates that the average ambient concentration of asbestos in the U.S is 1 f/m^3 in rural areas and 10 f/m^3 in urban areas Dr. Brodkin concedes that exposure to ambient levels does not increase the risk of mesothelioma but then appears to assert that any exposure occupational exposure above ambient levels does increase the risk. What if a resident of a rural area is exposed to chrysotile from joint compound that results in total exposure that is less than the total ambient exposure received by an urban dweller? By asserting that such exposure increases the risk of mesothelioma, Dr. Brodkin is clearly contradicting himself.

FRAMEWORK FOR ATTRIBUTION OF RISK OF MESOTHELIOMA TO SPECIFIC EXPOSURES

86. From the brief discussion above, it is clear that mesothelioma, like every other cancer, can occur spontaneously as a natural consequence of basic biological processes, and that risk of the disease can be increased by exposure to amphibole and, possibly, chrysotile exposure, and also by exposure to ionizing radiation. Once mesothelioma has occurred, the question of interest is whether a specific exposure to asbestos was a substantial contributing factor in the disease. For infectious diseases, the attribution of cause to specific agents is straightforward. For example, the sole cause of tuberculosis is a specific bacterium. It is therefore clear that any case of tuberculosis can be attributed to infection by the tuberculosis bacillus. There could be some doubt regarding the specific strain of the organism that is responsible for a specific case. But, generally, this issue can be settled by the appropriate laboratory tests.

87. The question of attribution becomes much more complex when a disease can occur spontaneously and/or have multiple causes. Most chronic diseases, such as cardiovascular disease and cancer, including mesothelioma as discussed above, have a multifactorial etiology, so that it is not generally possible to attribute the disease to any specific environmental exposure or life-style factor. It may be possible, however, to

[16] Biologically relevant radiation doses are measured in Sieverts. This unit of measurement incorporates consideration of the relative biological effectiveness of various kinds of ionizing radiation. A milliSievert, 1 mSv, is one thousandth of a Sievert.

[17] Naturally occurring ionizing radiation increases with altitude.

apportion the probability of causation quantitatively to specific exposures. That is, it may be possible to estimate the probability that the specific exposure in question was a causal factor in the development of the disease. What is clear is that, in any specific individual, any single exposure must be considered within the framework of all the factors that could have contributed to disease in that individual, including the probability of spontaneous occurrence. For example, consider a 70 year-old individual who develops lung cancer after a 50 pack-year[18] smoking history. Suppose this individual was also exposed to second-hand smoke in the workplace. Although the second-hand smoke could have made some contribution to this individual's risk of lung cancer, the most important and significant contribution was made by his/her smoking habit. Consider a second individual, a non-smoker who develops lung cancer at age 70 and was exposed to second-hand smoke in the workplace during his/her entire working life. In this case, second-hand smoke could have contributed substantially to the development of lung cancer. Thus, whether or not an exposure is a substantial contributing factor in disease causation depends on all other exposures received by the individual.

88. Attribution of cause has often been based on the notion of AF in epidemiology. Suppose that a disease has a certain spontaneous probability of occurrence and consider exposure to an agent that increases that probability. Consider now a population of individuals exposed to the agent and suppose that there are 100 cases of the disease in the population. What is the number of disease cases that can be attributed to the exposure? If we know the probability of disease among the non-exposed, we can compute the number of cases of disease in that population that would be expected spontaneously, *i.e.*, without any exposure. Suppose that number is 70. This is the number that would have occurred in the population anyway even without exposure. Then a total of 100-70 = 30 cases of the disease are attributable to exposure and the attributable fraction is 30/100 = 0.3. Formally, the AF is given by the formula:

$$AF = (P_E - P_0)/P_E = 1 - 1/RR = (RR - 1)/RR \qquad \text{Eqn. (1)}$$

where P_E is the probability of the disease among the exposed, P_0 is the probability of the disease among the unexposed, and $RR = P_E/P_0$ is the relative risk. The AF has often been interpreted as the probability that the disease in a diseased individual was caused by the exposure, and an attributable fraction of 0.5 or above, which corresponds to a RR of 2 or more, has been interpreted to mean that it was more likely than not that the exposure caused the disease. With more than one significant exposure, the situation is more complicated.

89. As I have stressed above, cancers are the end result of the accumulation of critical mutations in cells. These mutations can occur spontaneously during cell division without any exposure to a carcinogen. Therefore, cancers occur spontaneously, with the incidence of most cancers increasing with age. As described briefly above, environmental agents increase the probability of cancer in one of two ways, either by increasing the rates at which mutations occur or by increasing the net growth rate of

[18] This value corresponds to a smoking history of one pack of cigarettes per day for 50 years.

populations of cells that have acquired some of the mutations on the pathway to malignancy. Furthermore, multiple agents may act independently or in concert to increase the risk of cancer. Thus, estimating the contribution made by a specific exposure to total cancer risk in an individual requires knowledge of the entire exposure history of that individual to all environmental agents that could increase the risk of the cancer under question. Consider, for example, a male who develops mesothelioma at age 70 and who was exposed to crocidolite asbestos at a concentration of 2 f/ml between the ages of 25 and 40, to amosite at a concentration of 5 f/ml between the ages of 40 and 50, and was treated with radiation therapy for testicular cancer that had metastasized to the lung at age 50. Every one of these exposures could have contributed to his developing mesothelioma at age 70. In this example, however, only a fraction of this individual's risk of developing mesothelioma is attributable to his amosite exposure between the ages of 40 and 50. Consider, on the other hand, another male who develops mesothelioma at age 70 and is exposed to the same level of amosite (5 f/ml) between the ages of 40 and 50, but has no other exposures. In this second example, a much larger fraction of the individual's risk is attributable to his amosite exposure. This example illustrates that two individuals with mesothelioma may have identical exposures to amosite asbestos. In one of these individuals this exposure may be a substantial contributing factor to the mesothelioma, whereas in the other it might not. The role that other exposures played in the development of mesothelioma in these two individuals determines whether or not amosite exposure made a substantial contribution.

90. A proper analysis of the contribution made by each exposure to cancer risk often requires more information than is available. For the first individual in the example above, such an analysis would require good quantitative information on the following.

    a. The risk of spontaneous mesothelioma at age 70;

    b. The risk of mesothelioma at age 70 following exposure to crocidolite between the ages of 25 and 40;

    c. The risk of mesothelioma at age 70 following exposure to amosite between the ages of 40 and 50;

    d. The risk of mesothelioma following radiation therapy at age 50;

    e. Information on the interaction of these various exposures in causing mesothelioma.

91. In the second example above, the analysis is much simpler and requires only knowledge of the risk of spontaneous mesothelioma at age 70 and how this risk is modified by amosite exposure between the ages of 40 and 50. The crucial point that needs to be kept in mind here is that, although exposure to amosite is identical in the two hypothetical scenarios described above, this exposure contributed a much larger fraction of the risk of the mesothelioma in scenario 2 than in scenario 1. Thus, the fractional contribution made

by any exposure to asbestos to mesothelioma risk depends on the other exposures that the individual might have received.

92. The examples above deal with specific fiber types and the risk of mesothelioma. Similar considerations apply when attributing risk to specific occupations. For example, consider an individual who worked both as an insulator and a pipe-fitter. Both jobs, including the length of time spent in each job, should be considered in any estimation of the contribution to mesothelioma risk made by each occupation. Certain jobs involving work with friction products, such as automobile mechanics, have been shown not to increase the risk of mesothelioma in multiple epidemiologic studies. Such jobs make no contribution to the risk of mesothelioma whether or not the individual worked at other jobs associated with asbestos exposure (e.g. Hessel et al., 2004). A framework for the attribution of the risk of mesothelioma to specific asbestos exposures has been developed by Price and Ware (2005).

CONCLUSIONS

93. The evidence makes it abundantly clear that, like other cancers, mesothelioma can occur spontaneously without exposure to asbestos. Multiple, well-conducted epidemiological studies show that the risk of mesothelioma is not increased among automobile mechanics, suggesting strongly that low level exposures to chrysotile do not increase the risk of mesothelioma. The contention that small exposures over background contribute substantially to the development of mesothelioma is simply not supported by the science.

94. As I have discussed above, the only way to determine whether a specific exposure to asbestos was a factor in causing a plaintiff's disease is to conduct an explicit evaluation of the role of that asbestos exposure in causing the disease relative to the risk of developing the disease spontaneously and the additional risks imposed by other exposures, including other asbestos exposures and ionizing radiation. Such an evaluation involves estimating the additional risk, if any, imposed by the exposure at issue after taking into account the probability that the disease occurred spontaneously and the probability that other exposures, including other asbestos exposures and ionizing radiation, caused the disease. The contribution to risk made by any specific exposure will depend on how that exposure fits into the general pattern of exposure to asbestos and ionizing radiation for that individual. While detailed information is not always available, at the very least the total exposure, and the type of the asbestos fibers to which exposure occurred, need to be considered. If there are no other exposures to either asbestos or ionizing radiation, then the role of the exposure of interest must be evaluated within the framework of spontaneous occurrence of mesothelioma. When making this evaluation, it must be kept in mind that there is excellent affirmative epidemiologic evidence that exposure to low levels of chrysotile asbestos does not increase the risk of mesothelioma. Furthermore, there is little direct epidemiologic evidence that exposure to low levels of any kind of asbestos increases the risk of mesothelioma.

95. I have been asked to evaluate the methodology used by Dr. Brodkin to assess causation in this matter. In his notes and deposition in this matter, Dr. Brodkin identifies the following exposure sources related to Mr. Quirin: 1) employment in the U.S. Navy, 2) work as a telephone line installer at various construction sites, 3) smoking Kent cigarettes with micronite filters. Taking Dr. Brodkin's assessment as true, Mr. Quirin probably received substantial exposure to amphibole asbestos from all three.

96. There is compelling evidence that the U.S. Navy used substantial quantities of amphibole asbestos (Navy Bureau of Ships, [NBS] 1959, 1962; Rushworth, 2005; Franke and Paustenbach, 2011). In addition, numerous epidemiology studies have reported increased risk of mesothelioma associated with Navy and shipyard work (e.g., Blot and Fraumeni, 1981; Kolonel et al., 1985; Newhouse et al., 1985; Muscat and Wynder, 1991; Danielsen et al., 1993, 2000; Puntoni et al., 2001; Pan et al., 2005; Krstev et al., 2007). The scientific evidence establishes the risk, not just for the individuals handling the asbestos materials in shipyards or aboard ship, but also for those working in the vicinity (e.g., Selikoff et al., 1964; Kolonel et al., 1985; Danielsen et al., 1993; Puntoni et al., 2001). Amphibole asbestos has also been used in the construction industry and construction workers are known to be at increased risk of mesothelioma (Fletcher et al., 1993; Coggon et al., 1995; Robinson et al., 1996; Burnett et al., 1997; Wang et al., 1999; Hemminki and Li, 2003; Koskinen et al., 2003; NIOSH, 2003; McElvenny et al., 2005; Rake et al.; 2009). In addition, Mr. Quirin allegedly smoked Kent cigarettes with a micronite filter during a period when these filters could have contained crocidolite asbestos, the most potent asbestos fiber. Mr. Quirin's amphibole exposures, along with his age, are sufficient to have caused his mesothelioma.

97. Dr. Brodkin opines, however, that in addition to these very substantial exposures to amphibole asbestos, Mr. Quirin's bystander exposure to chrysotile asbestos from joint compound also contributed substantially to the development of his mesothelioma. Clearly, Dr. Brodkin has not critically evaluated whether Mr. Quirin's alleged bystander exposure to chrysotile asbestos added to the substantial risk imposed by his total amphibole exposure. Dr. Brodkin offers not a shred of evidence that chrysotile asbestos from joint compound increases the risk of mesothelioma.[19] He appears to base this conclusion on the assumption that any exposure over background to any kind of asbestos fiber increases the risk of mesothelioma. He ignores the large body of epidemiologic literature showing that exposure to low levels of chrysotile asbestos does not increase the risk of mesothelioma and does not add to the risk imposed by other exposures. He relies

[19] If Dr. Brodkin wants to contend that exposure to joint compound increases the risk of mesothelioma, then he must cite properly conducted analytic epidemiology studies showing that work with joint compound increases the risk of mesothelioma. It is not sufficient to cite the construction literature because construction workers are exposed to asbestos, and particularly amphibole asbestos, from various sources. If Dr. Brodkin wants to make the argument that joint compound is mesotheliogenic because of exposure to chrysotile, then he must be able to demonstrate that cumulative exposure from joint compound is in the several hundred fibers/ml-year range, because increased risk in predominantly chrysotile cohorts is seen only with such high exposures. Dr. Brodkin claims that he took the dose into account but, in reality, he has no quantitative estimate of the exposure that Mr. Quirin received from joint compound.

instead on case reports and on epidemiologic studies of commercial chrysotile contaminated with amphibole and involving very large exposures in the range of several hundred f/ml-year. He concludes from these very high exposure studies of chrysotile contaminated with amphibole that exposure to much lower levels of pure chrysotile increases the risk of mesothelioma. This conclusion is scientifically untenable.

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Appendix 1. Curriculum Vitae of Suresh Moolgavkar

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Corporate Vice President and Director of the Center for Epidemiology, Biostatistics, and Computational Biology

Professional Profile

Dr. Suresh Moolgavkar has more than 30 years of experience in the fields of epidemiology, biostatistics, and quantitative risk assessment. He is internationally known for his work in developing mechanistically based dose-response models for carcinogenesis, and, in particular, for the two-mutation clonal expansion model, also known as the Moolgavkar-Venzon-Knudson (MVK) model. For the past decade, Dr. Moolgavkar has also been keenly interested in air pollution epidemiology. Dr. Moolgavkar retired from his position as a Full Member of the Fred Hutchinson Cancer Research Center in August 2008. He continues to be an Affiliate Investigator at the Center and Professor of Epidemiology and Adjunct Professor of Applied Mathematics at the University of Washington in Seattle. Dr. Moolgavkar has served on the faculties of Johns Hopkins University, Indiana University, University of Pennsylvania, and Fox Chase Cancer Center. He has been a visiting scientist at the Radiation Effects Research Foundation in Hiroshima, the International Agency for Research on Cancer in Lyon, and the German Cancer Research Center in Heidelberg. Dr. Moolgavkar has served on numerous review panels and as a consultant to the National Cancer Institute, EPA, Health and Welfare, Canada, The International Agency for Research on Cancer (IARC), the California Air Resources Board (CARB), and the CIIT Centers for Health Research, among others.

Dr. Moolgavkar is the author or co-author of more than 160 papers and contributed chapters in the areas of epidemiology, biostatistics, and quantitative risk assessment, and has edited three books in these areas. He was the senior editor of a monograph, *Quantitative Estimation and Prediction of Human Cancer Risk*, published by the International Agency for Research on Cancer. He is an elected member of the American Epidemiological Society. Dr. Moolgavkar has served on the editorial boards of *Genetic Epidemiology* and *Inhalation Toxicology*, and is currently Associate Editor for Health and Environment of *Risk Analysis—An International Journal.* Dr. Moolgavkar has published numerous epidemiological and toxicological papers on lung cancer, including lung cancer following radiation and exposure to fibers. Dr. Moolgavkar was a member of the working group involved in the writing of the IARC monograph on tobacco smoking in 1986 (IARC monograph 38).

Dr. Moolgavkar was given the Founders' Award by the CIIT Centers for Health Research in 1990 and the Distinguished Achievement Award by the Society for Risk Analysis in 2001. He is an elected member of the American Epidemiological Society and a Fellow of the Society for Risk Analysis, the premier international organization for risk assessment.

Dr. Moolgavkar's research has been supported largely by grants from the National Institutes of Health, the U.S. Department of Energy, and EPA.

Academic Credentials and Professional Honors

Senior Fellow, Department of Epidemiology, University of Washington, 1976–1977
Ph.D., Mathematics, Johns Hopkins University, Baltimore, Maryland, 1973
Postdoctoral Fellow, Departments of Pharmacology and Biophysics, Johns Hopkins Medical School, Baltimore, Maryland, 1966–1968
M.B.B.S., (M.D.) Bombay University, 1965

Elected Member, American Epidemiological Society
Distinguished Achievement Award, Society for Risk Analysis, 2001
Founders' Award, Chemical Industry Institute of Toxicology, 1990
Lester R. Ford Award of Mathematical Association of America, 1977
Faculty Research Fellowship of Indiana University, 1974–1976

Academic Appointments

Professor, Department of Epidemiology, University of Washington, 1984–present
Adjunct Professor, Department of Biostatistics, University of Washington, 1984–2009
Adjunct Professor, Department of Applied Mathematics, University of Washington, 2004–present
Member, The Fred Hutchinson Cancer Research Center, Seattle, 1984–2008
Affiliate Investigator, The Fred Hutchinson Cancer Research Center, 2008–present
Member, Graduate Faculty, University of Washington, 1984–present
Adjunct Associate Professor, Department of Research Medicine, University of Pennsylvania School of Medicine, 1980–1984
Research Physician, The Institute for Cancer Research, Fox Chase Cancer Center, Philadelphia, 1979–1984
Clinical Assistant Professor, Department of Research Medicine, University of Pennsylvania School of Medicine, 1977–1980
Associate, American Oncologic Hospital, Philadelphia, 1977–1984
Epidemiologist, The Fox Chase Cancer Center, Philadelphia, 1977–1984
Member, Graduate Group in Epidemiology, University of Pennsylvania, 1977–1984
Assistant Professor of Mathematics, Indiana University, Bloomington, 1973–1977
Instructor in Mathematics, Johns Hopkins University, 1972–1973

Editorships and Editorial Review Boards

Editorial Board, *Inhalation Toxicology*, 2006–2008
Guest Editor, *Modeling and Data Analysis in Cancer Studies*, special issue of Mathematical and Computer Modelling, 33(12–13), 2001
Associate Editor, *Risk Analysis—An International Journal*, 2000–present
Senior Editor, *Risk Analysis – An International Journal* , special issue on impact of reduced tobacco smoking on lung cancer mortality in the U.S., 1975-2000, To appear, 2012
Senior Editor, *Quantitative Estimation and Prediction of Human Cancer Risk*, International Agency for Research on Cancer, Scientific Publications 131, 1999
Editor, *Scientific Issues in Quantitative Cancer Risk Assessment*, Birkhauser, Boston, 1990

Co-Editor, *Modern Statistical Methods in Chronic Disease Epidemiology*, John Wiley, 1986
Editorial Board, *Genetic Epidemiology*, 1984–1988

Publications

Mathematical

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Biomedical

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Meza R, Jeon J, Moolgavkar SH. Quantitative cancer risk assessment of nongenotoxic carcinogens. In: Cancer Risk Assessment: Chemical Carcinogenesis, Hazard Evaluation, and Risk Quantification. New York, Joh, Wiley & Sons, 2010.

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Moolgavkar SH. Comments on papers on U-shaped dose-response relationships for carcinogens. Hum Exper Toxicol 1998; 17:708–710.

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Tobacco Smoking. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. IARC, Volume 38, Lyon, 1986 (member of the working group).

Selected Invited Presentations

Moolgavkar SH. False discoveries: Challenges for understanding the environment. AAAS annual meeting, San Diego, February 2010.

Moolgavkar SH. Effects of education and primary prevention on lung cancer mortality trends. Erasmus University, Rotterdam, June 2009.

Moolgavkar SH. Multistage carcinogenesis and epidemiologic studies of cancer. University of Rochester Symposium in honor of Professor Andrei Yakovlev, April 2009.

Moolgavkar SH. Clonal expansion and carcinogenesis. International Conference on Systems Biology in Radiation Carcinogenesis, Munich, Germany, February 2007.

Moolgavkar SH. Epidemiology of colon cancer. AEK Cancer Congress, Frankfurt, Germany, February 2007.

Moolgavkar SH. Multistage carcinogenesis and epidemiologic studies of cancer. Distinguished Seminar Series, Fox Chase Cancer Center, PA, October 2005.

Moolgavkar SH. Multistage carcinogenesis and lung cancer prevention. IARC Seminar Series, Lyon, France, July 2004.

Moolgavkar SH. Radiation-induced gestational mutations and cancer. COSPAR meeting, Paris, France, July 2004.

Moolgavkar SH. Multistage carcinogenesis and radiation risk assessment. International Congress of Radiation Research, Brisbane, Australia, August 2003.

Moolgavkar SH. Cancer models and risk assessment. Environmental Mutagen Society, Annual Meeting, Miami, May 2003.

Moolgavkar SH. Methodological issues in time-series analyses of air pollution data. Meeting the Environmental Challenge of the 21st Century, Monterey, CA, March 2003.

Moolgavkar SH. Multistage carcinogenesis and risk assessment. International Biometrics Conference, Homburg, Germany, March 2001.

Moolgavkar SH. Multistage models of carcinogenesis: historical perspective, overview, implications for radiation carcinogenesis. International Workshop on Mathematical Models in Radiation Carcinogenesis, Kyoto, March 2001.

Moolgavkar SH. Modeling altered hepatic foci: issues and outstanding problems. 6th European Meeting on Hepatocarcinogenesis, Vienna, September 1999.

Moolgavkar SH. Intermediate lesions in carcinogenesis. Netherlands Institute for Health and the Environment Seminar Series, 1997.

Moolgavkar SH. Multistage model for lung cancer. International meeting of the Bernoulli Society, Calcutta, India, 1997.

Moolgavkar SH. Stochastic cancer models: Application to analyses of solid cancer incidence in the cohort of A-bomb survivors. Keynote Speaker, International symposium on low-dose and low-dose-rate radiation, Stratford-on-Avon, UK, 1997.

Moolgavkar SH. Stochastic models for estimation and prediction of cancer risk. International Symposium on Statistics in the Environment, Enschede, The Netherlands, 1997.

Moolgavkar SH. Time-series analyses of air pollution data. International Symposium on Health Effects of Particulate Air Pollution, Prague, 1997.

Moolgavkar SH. Multistage carcinogenesis, benzene exposure and leukemia risk. Berkeley Symposium on Benzene and Leukemia, Napa Valley, 1996.

Moolgavkar SH. Mutations and cell proliferation in cancer risk assessment. AACR International Workshop on Risk Assessment, Whistler BC, 1994.

Moolgavkar SH. Analysis of altered foci in rodent hepatocarcinogenesis experiments. European Toxicology Meeting, Mainz, Germany, 1993.

Moolgavkar SH. Biologically-based cancer risk assessment. International Symposium on Quantitative Risk Assessment, Research Triangle Park, NC, 1993.

Moolgavkar SH. Analysis of altered foci in rodent hepatocarcinogenesis experiments. International Workshop on Mouse Liver Tumors, Washington DC, 1992.

Moolgavkar SH. Cancer models and low-dose extrapolation of risk. Workshop on Risk Assessment and Low Dose Extrapolation, Zurich, Switzerland, 1992.

Moolgavkar SH. Cell proliferation and carcinogenesis. International Conference on Cell Proliferation in Carcinogenesis, NIEHS, North Carolina, 1992.

Moolgavkar SH. Multistage carcinogenesis and risk assessment. International Toxicology Conference, Rome, Italy, 1992.

Moolgavkar SH. A population perspective on multistage carcinogenesis. Princess Takamatsu Cancer Congress, Tokyo, Japan, 1991.

Moolgavkar SH: Cancer models. International Workshop on Biophysical Modelling of Radiation Carcinogenesis, Padua, Italy, 1991.

Moolgavkar SH. Carcinogenesis models: An overview. Hanford Symposium on Health and the Environment, Battelle PNL, Richland, WA, October 1990.

Moolgavkar SH. Analyses of altered foci in rat hepatocarcinogenesis experiments. University of Vienna Cancer Center, Vienna, Austria, July 1990.

Moolgavkar SH. Multistage models of carcinogenesis. University of Tübingen Seminar Series, Tübingen, July 1990.

Moolgavkar SH. Analyses of intermediate lesions in experimental carcinogenesis. German Cancer Research Center, Heidelberg, Germany, June 1990.

Moolgavkar SH. Analyses of altered foci in rat hepatocarcinogenesis experiments. BASF, Toxicology Group, Mannheim, 1990.

Moolgavkar SH. Cell proliferation and carcinogenesis. International Cancer Congress, Hamburg, 1990.

Moolgavkar SH. Multistage carcinogenesis. University of Pittsburgh, Department of Biostatistics Seminar Series, 1990.

Moolgavkar SH. Analysis of altered foci in hepatocarcinogenesis experiments. McArdle Laboratory, University of Wisconsin, Madison, WI, 1989.

Moolgavkar SH. Biologically-based cancer risk assessment. Society for Risk Analysis, Annual Meeting, San Francisco, CA, 1989.

Moolgavkar SH. Multistage carcinogenesis and radiation risk assessment. Radiation Research Society, Annual Meeting, Seattle, WA, 1989.

Moolgavkar SH. The role of somatic mutations and cell replication kinetics in quantitative cancer risk assessment. International Conference on Chemically Induced Cell Proliferation: Implications for Risk Assessment, Austin, TX, 1989.

Moolgavkar SH. Two mutation model for carcinogenesis: Relative roles of somatic mutations and cell proliferation in determining risk. SIMS Conference on Scientific Issues in Quantitative Cancer Risk Assessment, Alta, Utah, 1989.

Moolgavkar SH. Cancer models and risk assessment. NATO Workshop on Biologically-based Methods for Cancer Risk Assessment, Corfu, Greece, June 1988.

Moolgavkar SH. A two-stage model for carcinogenesis and its implications for risk assessment. University of Nebraska Medical Center, May 1988.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Risk Assessment Workshop, Washington, DC, March 1988.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Health and Welfare, Ottawa, Canada, March 1988.

Moolgavkar SH. Curvature and inference in exponential families: Application to Relative Risk Regression Models. Carleton University, Ottawa, Canada, March 1988.

Moolgavkar SH. Cox regression for the innocent bystander. Fox Chase Cancer Center Seminar, Philadelphia, PA, December 1987.

Moolgavkar SH, Prentice R. Modern statistical methods in chronic disease epidemiology. Biopharmaceutical Section of ASA (tutorial and short course), Newark, NJ, December 1987.

Moolgavkar SH. Biologically motivated two-stage model for carcinogenesis. 17th Conference on Toxicology, Wright-Patterson Air Force Base, Dayton, OH, November 1987.

Moolgavkar SH. Two-stage model for carcinogenesis. University of Wisconsin Seminars, "Curvature and Inference in Exponential Families: Application to Relative Risk Regression Models," Department of Human Oncology, Madison, OH, November 1987.

Moolgavkar SH. Two mutation model for cancer risk assessment. EPA Toxicology and Microbiology Seminar Series, Cincinnati, OH, October 1987.

Moolgavkar SH. Origin invariant relative risk functions: multi-stage models for cancer risk assessment. American Statistical Association Annual Meeting, San Francisco, CA, August 1987.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Risk Assessment Workshop, Washington, DC, March 1987.

Moolgavkar SH. Two-stage model for carcinogenesis: implications for risk assessment. Symposium on Quantitative Assessment of Cancer Risk, Washington, DC, February 1987.

Moolgavkar SH. A cohort analysis of smoking and cancers of the lung, bladder and pancreas. School of Public Health grand rounds, Department of Biostatistics Seminar on General Relative Risk Regression Models for Epidemiologic Studies, University of Pittsburgh, Pittsburgh, PA, January 1987.

Moolgavkar SH. Two-stage model for carcinogenesis and the IPI protocol. Battelle PNL, Richland, WA, 1986.

Moolgavkar SH. Modern statistical methods in chronic disease epidemiology. SIMS conference, Alta, UT, June 1985.

Moolgavkar SH. Time related factors in cancer epidemiology. NIH International Symposium, April 1985.

Moolgavkar SH. General relative risk models for case-control studies. Johns Hopkins University, School of Public Health, Baltimore, MD, 1985.

Moolgavkar SH. Stochastic models for carcinogenesis and risk assessment. EPA, Washington, DC, 1985.

Moolgavkar SH. Risk assessment using vital data. SIMS Conference on Environmental Epidemiology and Risk Assessment, Alta, UT, June 1982.

Selected Professional Activities

- Consultant, Fox Chase Cancer Center
- Consultant, Health and Welfare, Canada
- Consultant, University of Nebraska Medical Center
- Member, IARC (International Agency for Research on Cancer) working group on Tobacco Smoking
- Member, NIH Special Study Section for Biometry
- Member, NSF panel to review scientific bases of risk assessment methodologies
- Member, External Science Advisory Board, RISC-RAD project of the European Union, ongoing
- Member, External Science Advisory Board, California Air Resources Board, ongoing
- Invited Expert, Workshop on Mechanisms of Fiber Carcinogenesis, IARC, Lyon, France, November, 2005
- Area Editor for Health and Environment, *Risk Analysis—An International Journal*, Jan 2000–present
- Senior Editor of monograph *Quantitative Estimation and Prediction of Cancer Risk* IARC Scientific Publications, No. 131, 1999
- Co-chairman, International Conference on Mathematical Models in Cancer, Park City, Utah, 1998
- Member, Health Effects Institute Expert Panel for re-analyses of critical air pollution studies, 1997–2000
- Member, Working Group on quantitative estimation and prediction of cancer risk, IARC, Lyon, 1993
- Member, Scientific Advisory Panel to the CIIT Centers for Health Research, 1992–2005
- Member, Scientific Advisory Panel to review the EPA Dioxin Health Assessment document, 1992
- Member, Scientific Advisory Panel to review Risk Assessment program of the National Center for Toxicologic Research, 1992
- Organizer and Chair, SIMS conference "Scientific Issues in Quantitative Cancer Risk Assessment", held in Snowbird, Utah, June 1989
- Member, Advisory Committee to review risk assessment program of Armstrong Laboratories, Wright-Patterson Air Force Base, 1987
- Member, External Scientific Committee to review the program of the Radiation Epidemiology Branch, NCI, 1987
- Co-chairman of SIMS conference "Modern Statistical Methods in Chronic Disease Epidemiology" held in Alta, Utah, in June 1985
- Session Chairman at International Symposium: "Time Related Factors in Cancer Epidemiology," held at NIH in April 1985

EXHIBIT 15

2010 WL 1266659
Only the Westlaw citation is currently available.

United States District Court,
S.D. Indiana,
Indianapolis Division.

U.S. AUTOMATIC SPRINKLER, CO., Plaintiff,
v.
The RELIABLE AUTOMATIC SPRINKLER CO., and Ferguson Fire & Fabrication, Inc., f/k/a the Clark Group, Inc., Defendants.

No. 1:07–cv–00944–SEB–TAB. | March 25, 2010.

Attorneys and Law Firms

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Opinion

ORDER ADDRESSING MOTIONS TO EXCLUDE

SARAH EVANS BARKER, District Judge.

***1** This cause is before the Court on Defendant, The Reliable Automatic Sprinkler Company's ("Reliable"), Motion to Exclude Testimony of George Langford, Ph.D [Docket No. 104], filed on July 10, 2009; Defendant Ferguson Fire & Fabrication, Inc.'s ("Ferguson") Motion to Exclude Testimony of Dr. George Langford for Trial [Docket No. 106], filed on July 10, 2009; and Ferguson's Motion to Exclude Testimony of Dr. George Langford for Summary Judgment [Docket No. 113], filed on July 20, 2009. Although filed separately, each of these motions seeks substantially the same result: the exclusion of Plaintiff's proffered expert testimony, on both admissibility and procedural grounds. For the reasons detailed in this entry, these motions are *GRANTED* in part and *DENIED* in part.

Background

Plaintiff U.S. Automatic Sprinkler, Co. ("USAS") installed a sprinkler system at a project in Greenwood, Indiana ("Greenwood Project") using sprinkler heads manufactured by and purchased from Reliable, as well as "female" weld-o-lets purchased from Ferguson. After this installation had been completed, leaks began to occur. Although these leaks were promptly fixed, soon thereafter leaks began to occur with increasing regularity. Eventually, USAS was forced to undo and reinstall each sprinkler head, a process that included the replacement of the original sealant with a more expensive sealant.

Because of the high costs incurred in connection with resolving this problem, USAS hired Dr. George Langford to test the sprinkler heads and weld-o-lets USAS had originally installed to determine whether, in his opinion, these products were correctly manufactured, and whether a defect in their manufacture was the possible proximate cause of the leaks that occurred at the Greenwood Project. With regard to the Reliable sprinkler heads, Dr. Langford concluded that, in some cases, the heads incorporated what he referred to as "drunken threads," which is a variation in the helix angle of the threads. He also determined that some of the weld-o-lets were "out-of-round," a physical irregularity that can affect the performance of the threads during installation.

Dr. Langford's conclusion, which Plaintiff proffered in its summary judgment briefing and intends to offer at trial, was that the leaking at the Greenwood Project occurred during installation and was caused by this combination of mechanical irregularities. According to Dr. Langford, when the "drunken" sprinkler heads were mated with the "out-of-round" weld-o-lets, gaps occurred that created the potential for the type of leaking experienced at the project.

Discussion

Defendants seek to exclude the expert testimony of Dr. Langford. The admissibility of expert testimony is governed by the framework set out in Federal Rule of Evidence 702 and *Daubert v. Merrell Dow Pharms. Inc.* 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993). Applying this framework, courts must undertake:

> a three-step analysis: the witness must be qualified "as an expert by knowledge, skill, experience, training, or education"; the expert's reasoning or methodology underlying the testimony must be scientifically reliable;

and the testimony must assist the trier of fact to understand the evidence or determine a fact in issue.

***2** *Ervin v. Johnson & Johnson, Inc.,* 492 F.3d 901, 904 (7th Cir.2007) (quoting Fed.R.Evid. 702); *see also Kumhoe Tire Co., Ltd. v. Carmichael,* 526 U.S. 137, 141, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999) (extending the *Daubert* admissibility framework to expert testimony in the social sciences). "The *Daubert* standard applies to all expert testimony, whether it relates to an area of traditional scientific competence or whether it is founded on engineering principles or other technical or specialized expertise." *Smith v. Ford Motor Co.,* 215 F.3d 713, 719 (7th Cir.2000) (citing *Kumho,* 536 U.S. at 141).

I. Dr. Langford's Qualifications

We begin by examining Dr. Langford's expertise to determine whether he is qualified to perform the calculations and arrive at the conclusions contained in his report. "A court should consider a proposed expert's full range of practical experience as well as academic or technical training when determining whether that expert is qualified to render an opinion in a given area." *Smith,* 215 F.3d at 718. The "scientific knowledge" contemplated by *Daubert* "connotes more than subjective belief or unsupported speculation." *Porter v. Whitehall Labs., Inc.,* 9 F.3d 607, 613–14 (7th Cir.1993). To suffice, the proffered expert's knowledge must have "a grounding in the methods and procedures of science." *Daubert,* 509 U.S. at 590.

Defendants do not substantially challenge Dr. Langford's qualifications to testify as to this opinion. Dr. Langford possesses an undergraduate degree in metallurgy from the Massachusetts Institute of Technology and a Doctor of Science degree also from MIT. He has more than thirty-five years of experience as a metallurgist and has investigated hundreds of material-related problems in the areas of corrosion, mechanical and dimensional analysis, physical, mechanical, and chemical metallurgy, microstructural analysis, and optical and electron microscopy. He has consulted and testified in more than fifty cases involving insurance claims in the fire sprinkler industry. Dep. Of Langford at 8, 75. Based on this experience and knowledge, we find that Dr. Langford is fully qualified to testify as an expert on the issues presented in this case.

II. The Reliability and Helpfulness of Dr. Langford's Testimony

Even a "supremely qualified expert cannot waltz into the courtroom and render opinions unless those opinions are based on some recognized scientific method and are reliable and relevant under the test set forth by the Supreme Court in *Daubert." Clark v. Takata Corp.,* 192 F.3d 750, 759 n. 5 (7th Cir.1999). The testimony of a "well credentialed expert who employs an undisclosed methodology" or who offers opinions lacking "analytically sound bases" must be excluded. *Tuf Racing Products, Inc. v. American Suzuki Motor Corp.,* 223 F.3d 585, 591 (7th Cir.2000). Thus, although the Court's role does not include an assessment of the credibility or persuasiveness of the proffered testimony, which factual issues are left for the jury to determine, *Deputy v. Lehman Brother's, Inc.,* 345 F.3d 494, 506 (7th Cir.2003), the Court, "in its role as a gate-keeper," must nonetheless determine if Dr. Langford's opinions are based on reliable methodology, and whether they would be helpful to a jury. *Winters v. Fru–Con, Inc.,* 498 F.3d 734, 743 (7th Cir.2007).

***3** Defendants challenge Dr. Langford's methodology on the following grounds: (1) that he did not properly "test" his method; (2) that he sampled too few outlets to form a reasonably accurate opinion; (3) that his measuring methods were novel and unproven; (4) that his measuring equipment was tainted; (5) that he engaged in speculation rather than calculation at more than one juncture in his analysis; and (6) that his testimony is, at certain points, internally contradictory. Defendants' briefing, which is extensive, mounts numerous cogent and robust challenges to the accuracy and worthiness of Dr. Langford's methodology and conclusions. Among other specific challenges, Defendants point out that Dr. Langford made questionable assumptions, [1] failed to quantify numerous data that he collected, [2] failed to account for certain possible alternative explanations, [3] and failed to account for the different metallurgical properties of the components at issue.

Notwithstanding the force of these arguments, we find that by and large they go to the *weight* of the evidence Dr. Langford offers, rather than the admissibility of that evidence. "The trial court's gatekeeper role ... is not meant to supplant the adversary system, or the role of the jury: '[V]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof, are the traditional and appropriate means of attacking shaky, but admissible evidence." *United States v. Grace,* 455 F.Supp.2d 1148, 1153 (D.Mont.2006) (quoting *Daubert,* 509 U.S. at 596). In essence, Defendants seek exclusion by subjecting Dr. Langford to a contest with the opinions offered by their expert

and with hypothetical alternative methods and explanations for the facts presented in the case. This is not an appropriate approach to the *Daubert* inquiry.

Significantly, Dr. Langford subjected the evidence before him to multiple tests prior to arriving at the conclusions contained in his report. Whether the expert has subjected his theory to testing has been recognized as the most important reliability factor. *Chapman v. Maytag Corp.,* 297 F.3d 682, 688 (7th Cir.2002). In developing his testing methods, Dr. Langford clearly reviewed relevant studies related to similar experimentation undertaken by others in the field. Dep. of Langford at 142.[4] Furthermore, as part of the process he undertook to test the data, Dr. Langford employed various scientific controls, established an error rate, and repeatedly tested allegedly defective components to verify his results. Dep. of Langford at 51, 151, 155. Whether additional or alternative testing would undercut or support his testimony is a question of the weight to be given his conclusions, which shall be addressed at summary judgment or at trial. *See Marvin Lumber v. PPG,* 401 F.3d 901, 916 (8th Cir.2005).

As acknowledged by Defendants' expert, the methodologies that Dr. Langford referenced in designing his method are accepted and recognized in the relevant scientific community. For all of these reasons, we conclude that Dr. Langford evaluated the data before him with a sufficiently reliable methodology to satisfy the standards outlined in *Daubert.*

***4** We also find that Dr. Langford's testimony will assist the trier of fact. Dr. Langford was the only person, expert or otherwise, to conduct a detailed examination of the sprinkler heads and weld-o-lets that are the subject of Plaintiff's claims in the case at bar. His expertise, coupled with his substantial personal knowledge, indicate that his proffered testimony will be helpful to the resolution of the factual issues at stake.

Dr. Langford's educational background, experience in conducting tests similar to those that formed the basis of his report, and the reliability of his method allow us to conclude with relatively little difficulty that his expert report and testimony are admissible under *Daubert* requirements. Defendants' extensive challenges to his methods are more appropriately adduced at the summary judgment phase of the proceedings; the Court will resolve those issues at that time. For all of the foregoing reasons, Defendants' motions to exclude, insofar as the relate to the standards set forth in *Daubert* and Rule 702, shall be *denied.*

III. Dr. Langford's Newly Offered Affidavit

Defendants also contend that Dr. Langford's recent affidavits (Docket Nos. 102–9, 103–9, 119–8, 120–8) contain conclusions and opinions that must be excluded because they were not contained within his original report and were filed outside the deadlines established by the Court. District courts are empowered with broad discretion to set and enforce deadlines, including those established for the disclosure of expert witness testimony. *See, e.g., Bevolo v. Carter,* 447 F.3d 979, 981 (7th Cir.2006). According to Defendants, Dr. Langford's newly filed affidavits constitute an impermissible supplementation of Plaintiff's expert disclosures because those affidavits offer opinion testimony that Plaintiff was required to disclose in the original expert report. Pursuant to Federal Rule of Civil Procedure 26(a), which governs the circumstances under which a party may offer such supplemental evidence, a party's right to file rebuttal and supplementary expert reports does not permissibly extend the disclosure deadlines or "give license to sandbag one's opponent with claims and issues which should have been included in the expert witness' report." *In re Ready–Mixed Concrete Antitrust Litig.,* 261 F.R.D. 154, 159 (S.D.Ind.2009).

Plaintiff rejoins that the late filing of these affidavits was justified because Defendants did not disclose that their expert would utilize the information upon which Dr. Langford's supplemental affidavits rely, referred to as the "ESI Report" as well as specific American National Standard Institute ("ANSI") standards. Plaintiff's argument is not, however, supported by the facts before the Court. From our review of the record, it is clear that Defendants discussed their use of this information in a timely fashion and placed Plaintiff on notice that this information would be used in the formulation of Defendants' expert evidence. Plaintiff and its expert were therefore responsible for being prepared to discuss the specific portions of the ANSI standards and the ESI Report in question. Plaintiff has provided no satisfactory reason for its failure to comply with the deadlines for the disclosure of expert witness testimony, as established by the Court's case management plan and Fed.R.Civ.P. 26. Permitting Plaintiff to sidestep its own error by now allowing Dr. Langford's additional, late-filed affidavits to be considered would unfairly prejudice Defendants. *See Musser v. Gentiva Health Services,* 356 F.3d 751, 757 (7th Cir.2004). Accordingly, Defendants' motions are *granted* to this extent, and the subject affidavits shall be stricken.

IV. Conclusion

***5** For the reasons detailed in this entry, Defendants' Motions to Exclude the Testimony of Dr. George Langford are *GRANTED* in part and *DENIED* in part.

IT IS SO ORDERED.

Parallel Citations

82 Fed. R. Evid. Serv. 102

Footnotes

1 The most prominent of these is his assumption that the outlet threads were perfect when originally manufactured by a non-party. According to Defendants, making such an assumption is not appropriate.

2 Specifically, Defendants contend that he failed to quantify the "drunkenness" and "out-ofroundness" of the allegedly faulty components as well as failed to statistically account for the role played by heat in the installation process.

3 Here, Defendants detail at length the opposing theories offered by their expert.

4 Dr. Langford documented his testing methods in detail, such that another expert with his notes at hand could replicate his work. Moreover, his methods reflected common approaches to problems such as those presented in this litigation. He took detailed measurements of the tapered threads in the sprinkler heads using tools such as a screw-cutting lathe with a taper attachment, dial indicators with a probe component, and a commercial data plotting program, all of which are commonly used in scientific measurements similar to those involved in the case at bar.